Giancarlo Vignoli

Urodynamics for Urogynecologists

A Pocket Guide for Clinical Practice

Springer

Urodynamics for Urogynecologists

Giancarlo Vignoli

Urodynamics for Urogynecologists

A Pocket Guide for Clinical Practice

Springer

Giancarlo Vignoli
Functional Urology Unit
Casa Madre Fortunata Toniolo
Bologna
Italy

ISBN 978-3-319-74004-1 ISBN 978-3-319-74005-8 (eBook)
https://doi.org/10.1007/978-3-319-74005-8

Library of Congress Control Number: 2018940775

Printed on acid-free paper

This Springer imprint is published by the registered company Springer International Publishing AG part of Springer Nature
The registered company address is: Gewerbestrasse 11, 6330 Cham, Switzerland

Preface

In recent years gynecologists have shown an increasing interest in lower urinary tract dysfunctions related to gynecologic disease, but many of them are unfamiliar with urodynamics. It is a common belief that urodynamics can reveal the underlying mechanisms of lower urinary tract symptoms and that this may help to select more rational treatments. Particularly when the clinical evaluation is uncertain, urodynamics can provide additional information that may help to define the diagnosis. For decades urodynamic studies have been considered the gold standard to objectively diagnose a dysfunction of the lower urinary tract. Unfortunately, the role of urodynamic studies in patients with lower urinary tract dysfunction is largely based on expert opinion and the conventional use of urodynamic testing in clinical practice failed to support this original assumption. The differentiation between several types of lower urinary tract dysfunctions and the choice of treatment according to urodynamics data doesn't seem to have significant predictive value in most of the patients.

Possible explanations for such unexpected conclusion have been summarized by Griffiths et al.*: first, symptoms of lower urinary tract have similar underlying pathophysiology requiring similar treatment regardless of urodynamic data; second, current urodynamic tests are too rough (or to poorly done) to address the important pathophysiological differences that affect treatment outcome; third (more distressing) current treatments are so nonspecific that the underlying dysfunction is unimportant: treatment work equally well or poorly in any case.

To make matters worse, urodynamic testing is time consuming, invasive, and costly and not without associated morbidity. Furthermore, since the quality of the studies is crucial, they also require special training.

Today, the pros and cons of performing routine urodynamic testing have become a topic embroiled in controversy.

Particularly among gynecologists, since with the introduction of easy to administer mid-urethral slings, a simplified reasoning has taken place that states that every type of SUI may be treated in the same way, and therefore, no urodynamic investigation would be needed.

Since 2006 the National Institute for Clinical Excellence (NICE) recommends that the use of multichannel urodynamics is not routinely needed before surgery in women with a clearly defined clinical diagnosis of pure SUI.

LUTS in women, however, are not only urinary incontinence, but include the overactive bladder syndrome, the emptying disorders, and painful bladder syndrome, all of which should require urodynamic investigation for a proper assessment.

Despite the debate, there is no doubt that urodynamic evaluation of lower urinary tract function is still the best available tool for lower urinary tract function assessment. The evidence for urodynamic testing of patients with lower urinary tract dysfunction is extensive and solid. Urodynamics is necessary because it contributes to knowledge of lower urinary tract dysfunction, whether or not there is a narrow evidence that it improves outcome. Although pragmatic treatment of symptoms is possible as a first approach, only urodynamic testing can disclose the function of the lower urinary tract and provide the rationale for the most appropriate therapy.

The objective of this book is to verify the role of urodynamics in current urogynecological practice and the opinion about the need to perform urodynamic investigation in the various clinical situations that are part of female urology.

Each chapter is structured according to a specific scheme including the background of the problem, the theoretical role

of urodynamics in clarifying the nature of the problem, and, finally, the pros and cons of the investigation according to literature review. The first part of the book is devoted to conducting a urodynamic investigation properly, the so-called "good urodynamic practice." Some recent surveys indicate that most of the personnel performing urodynamics believe that their training had been inadequate. Training and education raises the level of confidence and the ability to perform and interpret urodynamic investigations, with obvious implications for the accuracy, reliability, and consistency of the final report.

Finally, a chapter is devoted to neurogenic bladder in women, which requires special considerations that are not seen with many male neurogenic bladder patients.

Our purpose is to provide urogynecological practitioners with a practical, easy-to-read, approach to indications, performance, and analysis of urodynamic testing in the field of female urology.

Bologna, Italy Giancarlo Vignoli

**Griffiths DJ, Kondo A, Bauer S, Diamant N, Liao L, Lose G, et al. Chapter 11: Dynamic testing. In: Abrams P, Cardozo L, Khoury S, Wein A, editors. Incontinence, Volume 1: Basics and evaluation, 3rd ICI, Edition 21. Paris, France: Health Publication Ltd; 2005. pp 585–673.*

Contents

Abbreviations

ACC	Anterior cyngulate cortex
ALPP	Abdominal leak point pressure
AUA/SUFU	American Urological Association/Society of Urodynamics and Female Urology
AUM	Ambulatory urodynamic monitoring
BOO	Bladder outlet obstruction
BOOI	Bladder outlet obstruction index
BPS	Bladder painful syndrome
CLPP	Cough leak point pressure
CIC	Clean intermittent catheterization
DHIC	Detrusor hyperactivity with impaired contractility
DLPP	Detrusor leak point pressure
DO	Detrusor overactivity
DU	Detrusor underactivity
EMG	Electromyography
FDV	First desire to void
FSF	First sensation of filling
FVC	Frequency volume chart
GUP	Good urodynamic practice
HSB	Hypersensitive bladder
ICI	International Consultation on Incontinence
ICS	International Continence Society
ICI-RS	International Consultation on Incontinence Research Society
IDC	Involuntary detrusor contraction(s)
IDO	Idiopathic detrusor overactivity
ISD	Intrinsic sphincteric deficiency

LPP	Leak point pressure
LUT	Lower urinary tract
LUTD	Lower urinary tract dysfunction(s)
LUTS	Lower urinary tract symptoms
MCC	Maximum cystometric capacity
MS	Multiple sclerosis
MUCP	Maximum urethral closure pressure
MUP	Maximum urethral pressure
MUS	Mid-urethral sling
NDO	Neurogenic detrusor overactivity
NDV	Normal desire to void
NICE	National Institute for Health and Clinical Excellence
NLUTD-S	Neurogenic lower urinary tract dysfunction syndrome
NPV	Negative predictive value
OAB-S	Overactive bladder syndrome
PAG	Periaqueductal grey
PCR	Polymerase chain reaction
PBS/IC	Pain bladder syndrome/interstitial cystitis
PFC	Pre-frontal cortex
PFM	Pelvic floor muscles
PFMF	Pelvic floor muscles function
PFS	Pressure-flow study
PHC	Parahippocampal complex
PMC	Pontine micturition center
POP	Pelvic organ prolapse
POP-S	Pelvic organ prolapse syndrome
PPV	Positive predictive value
PVR	Post-void residual
QoL	Quality of life
SCI	Spinal cord injury
SNM	Sacral neuromodulation
SD	Standard deviation
SDV	Strong desire to void
SMA	Supplementary motor area
SUI-S	Stress urinary incontinence syndrome
UAB	Underactive bladder

UDS	Urodynamic studies
UPP	Urethral pressure profile/profilometry
USI	Urodynamic stress incontinence
UTI	Urinary tract infection
UUI	Urgency urinary incontinence
VLPP	Valsalva leak point pressure
VUD	Videourodynamic
VUR	Vesico-ureteric reflux
VD-S	Voiding dysfunction syndrome
V V	Voided volume
WMH	White matter hyperintensities

Chapter 1
Physiology of Micturition in Female

1.1 Background

The two major functions of lower urinary tract are the storage and emptying of urine. Since voiding takes between 1 and 2 min and is performed four or five times a day, the bladder is in the storage mode for most of the time of daily life (Fig. 1.1). Going to the toilet is an essential everyday event normally done without paying excessive attention to the context. However, in order to accomplish this apparently simple sequence of events, a whole range of complex interacting systems are involved including the generation of information from the lower urinary tract and its following processing in the spinal cord, brain stem, and forebrain.

Furthermore, whereas many other visceral functions (i.e., cardiovascular system, GI tract) are regulated involuntarily, micturition requires the integration of autonomic and somatic efferent mechanisms to coordinate the activity of visceral organs (the bladder and urethra) with that of urethral striated muscles.

1.1.1 Central Neural Mechanisms Controlling Storage and Voiding

An early attempt to provide a simple picture of neural control of micturition was made by Bradley who suggested that control was exerted by four neural circuits ("loops"). Loop I,

G. Vignoli, *Urodynamics for Urogynecologists*,
https://doi.org/10.1007/978-3-319-74005-8_1

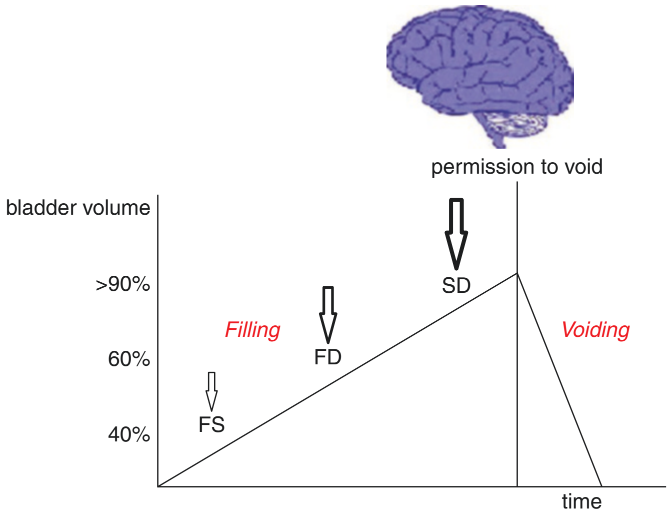


FIGURE 1.1 Micturition cycle: *FS* first sensation at 40% of bladder capacity, *FD* first desire at 60% of bladder capacity, *SD* strong desire at 90% of bladder capacity. Bladder sensation has a pivotal role in micturition cycle since the overall motivation of micturition is to attain relief from the increasingly intense sensation of bladder filling by emptying the bladder

consisting of connections between the brain stem and the frontal cortex, initiates and inhibits switching between filling and voiding states. Loop II, running spinally between the brain stem and sacral cord, is the main pathway of micturition reflex coordinating detrusor contraction and sphincter relaxation. Loops III and IV, connecting the sacral cord and bladder, are important in spinal reflexes that guarantee urethral tone at rest and proper coordination between the detrusor and sphincter during voiding. Loop V, from the motor cortex and Onuf's nucleus, is concerned with the voluntary control of the sphincters and pelvic floor muscles (Fig. 1.2).

Over the past 15 years, functional brain imaging has emerged as the most powerful technique for studying human brain function, in particular for understanding the relationship between activity in certain brain areas and

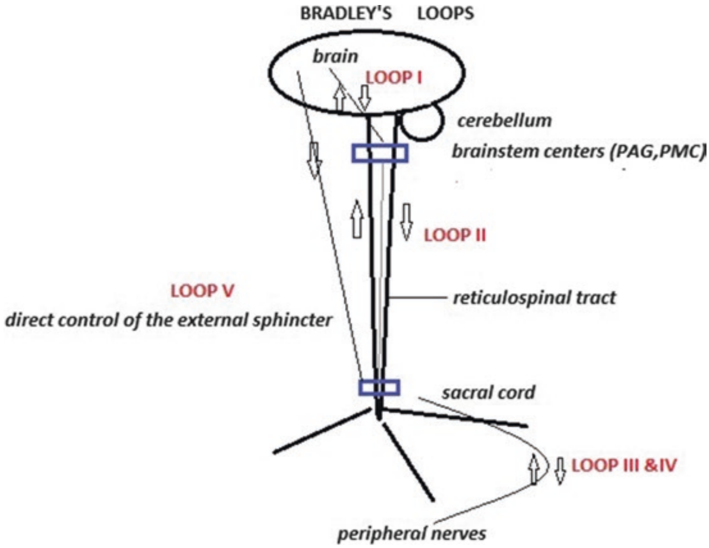


FIGURE 1.2 Neural control of micturition—Bradley loops: *Loop I* is a bidirectional pathway between the brain stem and cerebral cortex that includes also connections to subcortical nuclei (e.g., thalamus, basal ganglia, and amygdaloid nucleus). *Loop II* provides the brain stem control of the detrusor muscle. It is also a bidirectional pathway through the reticulospinal tract, including ascending impulses from the detrusor muscle to the brain stem micturition centers and descending impulses from the brain stem micturition centers to the sacral spinal cord. *Loops III* and *IV* provide for segmental control of pudendal pathways. *Loop V* provides for supraspinal control of striated muscles of periurethral area

specific functions. The micturition cycle is under control of specific neural networks which behave as a switch between storage of urine and voiding with the brain functioning as the master control of the entire process. Basically, voiding reflex can be broken into two parts: the long-loop, spinobulbospinal voiding and the forebrain circuits that modulate this reflex. The PAG (periaqueductal gray) acts as a pivotal region that distributes incoming signals from the bladder to many regions of the forebrain where they generate sensations

such as the desire to void and receive back signals from forebrain structures to decide whether or not the voiding reflex should be triggered. A provisional model of brain-bladder control has been developed in the last decade including three neural "circuits" which by handling bladder sensations can promote or delay the voiding reflex acting on the midbrain (Fig. 1.3).

- **Circuit 1** involves the PFC (prefrontal cortex), the PAG (periaqueductal gray), and PMC (pontine micturition center) and is responsible for voluntary initiation of micturition.
- **Circuit 2** involves the insula, the ACC (anterior cingulate cortex), and the adjacent SMA (supplementary motor area) and is mostly involved in handling of bladder sensation.
- **Circuit 3** involves subcortical regions such as the parahippocampal complex which might send safety or unsafety signals of voiding to pontine micturition center.

Storage phase occurs largely unconsciously and presumably relies on a subcortical network which maintains inhibition of the voiding reflex during bladder filling and monitors unusual or unexpected bladder events.

As the bladder fills, afferent signals increase in strength until they exceed a threshold set in the brain stem, specifically the midbrain periaqueductal gray (PAG).

PAG notifies the forebrain about bladder fullness through the activation of insula and ACC, and people perceive this signal as a strong desire to void or urgency (circuit 2). Activation of ACC is strictly related to activation of SMA that is known to tighten the pelvic floor muscles and the urethral sphincter, and this appears to be the mechanism to withholding urine. These informations are also passed to the prefrontal cortex (PFC) where the decision to void or not is made. If the decision is not to void, the PFC sends an inhibitory signal to the periaqueductal gray (PAG) and pontine micturition center (PMC) to inhibit the bladder to contracting until a bathroom is found. The ability of the brain to control the PMC is part of the social training that children

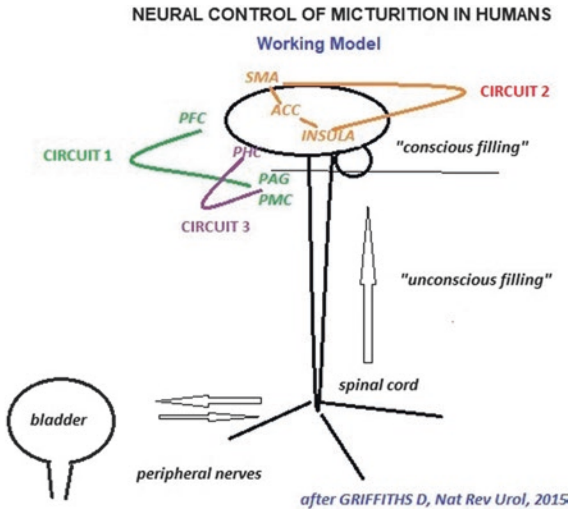


FIGURE 1.3 Neural control of micturition—Simple working model: three neural circuitry control lower urinary tract acting as a switch between storage of urine and voiding. As we know, a normal adult can postpone or advance the moment of micturition ensuring that urination occurs only if it is consciously desired and socially appropriate. During storage phase, afferent signals from the bladder are conveyed with increasing strength to the brain stem, specifically to the midbrain periaqueductal gray (PAG) where they remain silent until they exceed a threshold set. PAG afferents are mapped in the insula, forming the basis of sensation and are conveyed to the anterior cingulate cortex (ACC) which provides to inform the prefrontal cortex about the need of voiding (circuit 2). In addition, ACC is closely related to SMA. Activation of SMA is known to tighten the pelvic floor and urethral sphincter, and this appears to be a backup continence mechanism, used in critical situations to prevent incontinence. Circuit 3, including the parahippocampal region, tend to suppress voiding at PAG and might be part of a subcortical circuit which may send "safe" or "unsafe" signal to the pontine micturition center. Voluntary voiding is initiated by the medial prefrontal cortex which activates PAG and in turn PMC (circuit 1) .When activated the PMC passes on a signal to the sacral spinal cord where inhibitory interneurons in Onuf's nucleus relax the urethral sphincter at the same time as the bladder contracts ensuring a coordinated synergic voiding. Abbreviations: *PMC* pontine micturition center, *PAG* periaqueductal gray, *ACC* anterior cingulate cortex, *SMA* supplementary motor area, *PFC* prefrontal cortex, *PHC* parahippocampal complex

experience during growth and development. In infants, the brain is not mature enough to command the bladder, so the control of micturition comes from the signals sent from the spinal cord. When urine fills the infant bladder, an excitatory signal is sent to the sacral cord. When this signal is received by the sacral cord, the spinal reflex center automatically triggers the detrusor to contract. The result is an involuntary detrusor contraction with coordinated voiding.

In adults, when the decision to void is made, the PFC relaxes its inhibition of the PAG which, in turn, relaxes the PMC (circuit 1). The PMC sends descending motor output to the sacral spinal cord that ultimately relaxes the urethral sphincter and contracts the detrusor, so that voiding occurs. Voiding is continued to completion by continuing afferent input, probably to the PAG.

Relative to the other circuits, the role of circuit 3 is less known and still speculative. The parahippocampal complex is known to be involved in the retrieval of episodic memories and recognition of scenes and social context. Since it behaves like PFC, one might argue that it could send signals of "safety" or "unsafety" of micturition event to the PMC.

OAB, urgency incontinence, as well as chronic retention of urine may occur as a consequence of known cerebral lesions but often are idiopathic implying that there is a subtle lesion that may involve damage to cerebral connecting pathways or simply dysfunction of cortical and/or subcortical circuits capable of compromising the stability of voiding reflex.

The presence in some brain regions of white matter hyperintensities (WMH), which are structural abnormalities that appear with aging and may be linked to small vessel disease, is associated with LUT disorders (Fig. 1.4). The extent of white matter changes relates to functional brain activity during urgency. Activations in the posterior cortex and cerebellum are increased in those subjects with more intense white matter changes, while activity in the ACC and SMA is decreased. Deactivation in PFC is less pronounced, and the activity in this region increases with the increase of WMH. Such findings suggest that structural changes in white

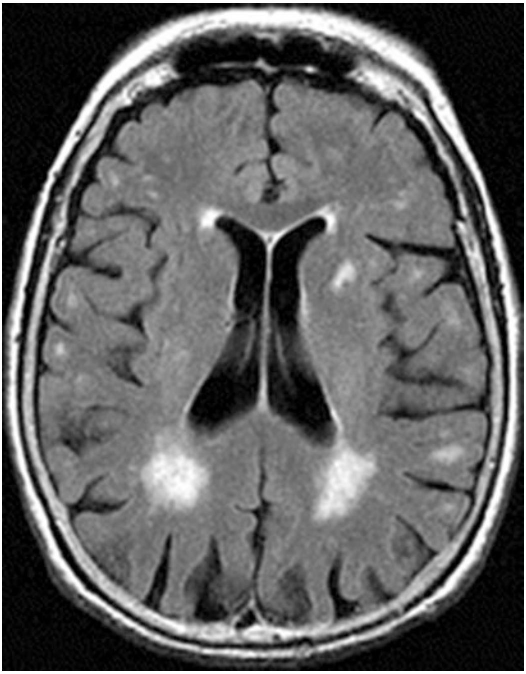


FIGURE 1.4 White matter hyperintensities in elderly women. Hyperintensities appear as "bright signals" (bright areas) on an MRI image

matter affect the regional brain activity involved in continence control, possibly making efforts to regulate less effectively. Structural damage of individual white matter pathways connecting these functionally important regions is most likely a mechanism of white matter effects on continence control.

Even emotions experienced in higher brain centers may exert downstream effects on the PMC, which is why some people can experience incontinence with excitement or fear.

In summary, regional brain activity differs in subjects with different functional and phenotypic characteristics. Such regions of difference may serve as potential markers for functional characterization of continence impairment. Imaging

methods, when coupled with urodynamic studies, may give an insight on neural correlates in clinical syndromes of impaired continence control (the Griffiths "new urodynamics").

1.1.2 Peripheral Neural Mechanisms Controlling Storage and Voiding

The control of the bladder provides a good example of the interplay between the voluntary somatic nervous system and the sympathetic and parasympathetic divisions of the autonomic nervous system, which operate involuntarily.

As shown in Fig. 1.5, the sympathetic innervation arises in the thoracolumbar outflow of the spinal cord (T11–L2), whereas the parasympathetic and somatic innervation originates in the sacral segments of the spinal cord (S2–S4). Afferent axons from the lower urinary tract also travel in these nerves.

Parasympathetic nervous fibers—via the pelvic nerve—release both cholinergic (ACh) and non-adrenergic, non-cholinergic transmitters. Cholinergic transmission is the major excitatory mechanism in the human bladder, resulting in detrusor contraction and consequent urinary flow. The effect is mediated principally by the M_3 muscarinic receptor, although bladder smooth muscle also expresses M_2 receptors.

Non-cholinergic excitatory transmission is mediated by ATP on P2X purinergic receptors in the detrusor muscle, whereas inhibitory input to the urethral smooth muscle is mediated by nitric oxide (NO).

Sympathetic nervous fibers—via the hypogastric nerve—release noradrenaline, which activates β3-adrenergic inhibitory receptors in the detrusor muscle to relax the bladder and α-adrenergic excitatory receptors in the urethra and the bladder neck.

Somatic cholinergic motor nerves supply the striated muscles of the external urethral sphincter. They arise in S2–S4 motor neurons in Onuf's nucleus and reach the periphery through the pudendal nerves. A medially placed motor

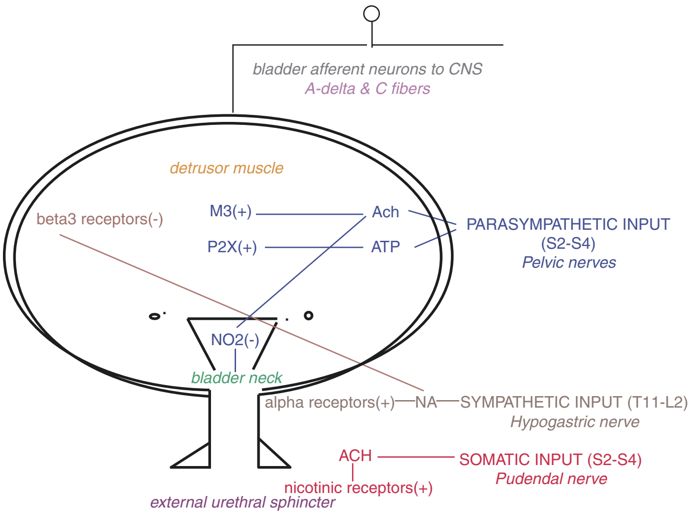


FIGURE 1.5 Peripheral bladder innervation: Parasympathetic pelvic nerve innervates the detrusor muscle. The parasympathetic neurotransmitter acetylcholine induces micturition by activating M3 receptors. In addition parasympathetic fibers release NO that relaxes actively the urethra. Sympathetic hypogastric nerve ensures continence. The sympathetic neurotransmitter norepinephrine inhibits the detrusor muscle by beta-receptors and increases the urethral tonus by alpha-receptors. Somatic pudendal nerve innervates the external urethral sphincter and pelvic floor muscles by releasing ACh at the motor plate. Sensations of the bladder are conveyed to CNS through A-delta fibers and C-fibers whose cell bodies are located in the dorsal root ganglia (DRG) at the level of S2–S4 and T11–L2 spinal segments

nucleus at the same spinal level supplies axons that innervate the pelvic floor musculature (Fig. 1.6).

Activation of the pudendal nerve causes contraction of the external sphincter, which occurs with activities such as Kegel exercises which also target the pelvic floor muscles. Some reflex activity may also occur through the pudendal nerve, such as the sphincter contractions that occur during sneezing, coughing, and laughing—the so-called guarding reflex.

Sensations of bladder fullness are conveyed to the spinal cord by the pelvic and hypogastric nerves, whereas sensory

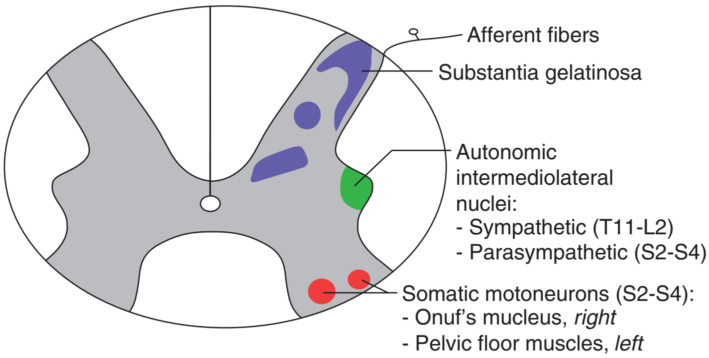


FIGURE 1.6 Organization of bladder motor nuclei (autonomic and somatic) in lateral and ventral horns of the spinal gray matter

input from the bladder neck and the urethra is carried in the pudendal and hypogastric nerves. The afferent components of these nerves consist of myelinated (Aδ) and unmyelinated (C) axons. The Aδ-fibers respond to passive distension and active contraction and thus convey information about bladder filling. The C-fibers are insensitive to bladder filling under physiological conditions (they are therefore termed "silent" C-fibers) and respond primarily to noxious stimuli such as chemical irritation or cooling.

1.1.3 Pressure Variations During Micturition Cycle

Bladder is a low-pressure reservoir. During filling phase, sensory afferent signals are carried via the pelvic and hypogastric nerves to the spinal cord, where they are relayed to the pontine micturition center and, in turn, to the periaqueductal gray via the lateral spinothalamic tracts and dorsal columns. Sympathetic tonus via the hypogastric nerve maintains smooth muscle-based activity of the urethral sphincter and aids in detrusor relaxation, which thus promotes urine storage keeping bladder pressure low with no phasic contractions and high compliance.

As afferent signaling increases in intensity with bladder filling, a threshold of consciousness is reached, at which point a socially appropriate opportunity to void is sought. During urine storage, there are a number of physiologic mechanisms to maintain continence. The external sphincter progressively contracts with bladder filling and will also contract during any sudden increase of abdominal pressure (i.e., during cough)—the so-called guarding reflex. During straining or Valsalva maneuver, there is equal transmission of pressure from the abdomen to the urethra (Fig. 1.7).

With permission to void, external sphincter relaxation is the first step in micturition event and precedes the detrusor contraction. Pontine signaling to the sacral cord results in relaxation of the striated muscle of the pelvic floor and parasympathetic cholinergic activation of the detrusor and which allows the voiding phase which can be defined a pressurized flow.

However, men and women have unique micturitional characteristics. What is normal voiding pressure and flow rate for men is not necessarily normal for women, since the voiding dynamics of women are different from those of men.

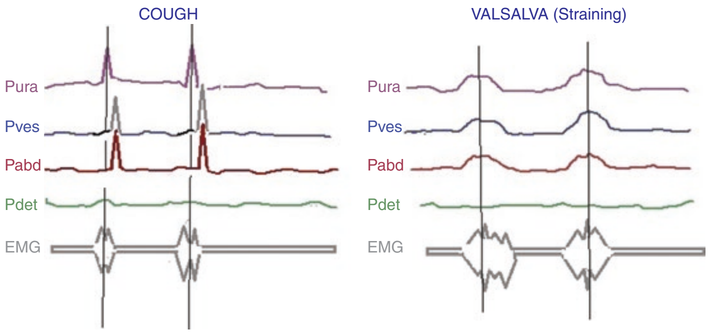


FIGURE 1.7 Pressure events during cough and straining (Valsalva maneuver). Immediately prior to cough there is a reflex contraction of the sphincter causing a rise in urethral pressure (Pura), while during straining or Valsalva maneuver, there is equal transmission of pressure from the abdomen to the urethra

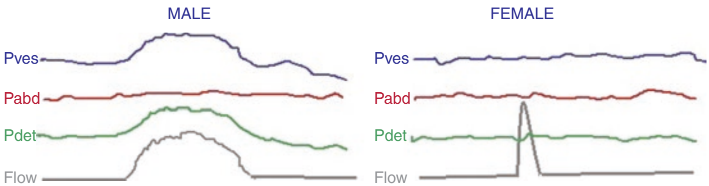


FIGURE 1.8 Voiding dynamic in men and women: unlike men, women may be able to void by prevalent relaxation of the pelvic floor muscles with lower contraction pressure or acontraction

In men a significant rise of detrusor pressure is recorded during voiding because the detrusor has to exert a force strong enough to move the urine through a relatively high-resistance conduit due to the presence of prostatic urethra.

Conversely, in normal women there is a very little increase in detrusor pressure during voiding, even when bladder contractility is normal, because the flow of urine encounters minimal resistance against female urethra (Fig. 1.8).

In addition many women void by pelvic floor relaxation rather than by detrusor contraction.

Valsalva-induced voiding across a relaxed pelvic floor is the third common voiding pattern seen in women (see Box 1.1) (Fig. 1.9):

During voluntary interruption of voiding or attempting to prevent voiding during an involuntary detrusor contraction, the external sphincter is contracted, stopping the stream, and

Box 1.1: Micturition Patterns in Women

- Pelvic floor relaxation (60%)
- Detrusor contraction (20%)
- Abdominal straining (20%)

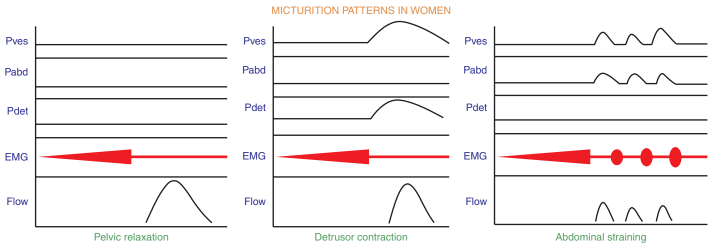


FIGURE 1.9 Micturition patterns in women

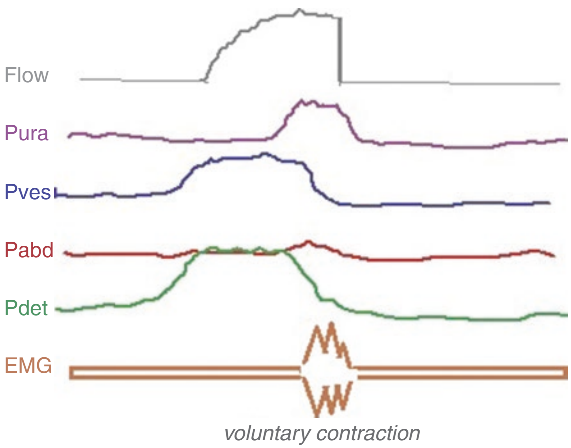


FIGURE 1.10 Voluntary interruption of voiding

then, through a reflex mechanism, the detrusor contraction, if present, decreases (Fig. 1.10).

Due to anatomical and behavior differences in micturition among men and women, one may expect regional brain activity to differ during bladder filling and voiding according to gender. To date, however, no significant differences in brain activation patterns between females and males have been reported.

In addition, the different voiding mechanisms do not lead to significant differences in parameters of the PFS and free uroflowmetry except detrusor contractility.

However, these voiding peculiarities are very important, since female patients unable to generate adequate detrusor pressure may be at higher risk for prolonged postoperative retention after anti-incontinence surgery and are less likely to be cured by intervention.

Suggested Reading

Neural Control of Micturition

Arya N, Weissbart S, Xu S, Rao H. Brain activation in response to bladder filling in healthy adults: an activation likelihood estimation meta-analysis of neuroimaging studies. Neurourol Urodyn. 2017;36:960–5.

Arya N, Weissbarth S. Central control of micturition in women: brain-bladder pathways in continence and urgency urinary incontinence. Clin Anat. 2017;30:373–84.

Smith PP, Kuchel GA, Griffiths D. Functional brain imaging and the neural basis of voiding dysfunction in older adults. Clin Geriatr Med. 2015;31:549–65.

deGroat WC, Griffiths D, Yoshimura N. Neural control of the lower urinary tract. Compr Physiol. 2015;5:327–96.

DeGroat WC, Yoshimura N. Anatomy and physiology of lower urinary tract. Handb Clin Neurol. 2015;130:61–108.

Bortolini MA, Bilhar AP, Castro RA. Neural control of lower urinary tract and targets for pharmacological therapy. Int Urogynecol J. 2014;25:1453–62.

DeGroat WC, Wickens C. Organization of the neural switching circuitry. Underlying reflex micturition. Acta Physiol (Oxford). 2013;207:66–84.

Tadic SD, Tannenbaum C, Resnick NM, Griffiths D. Brain responses to bladder filling in older women without urgency incontinence. Neurourol Urodyn. 2013;32:435–40.

Fowler CJ, Griffiths D, deGroat WC. The neural control of micturition. Nat Rev Neurosci. 2008;9:453–66.

Fowler CJ. Integrated control of lower urinary tract—clinical perspective. Br J Pharmacol. 2006;147:S14–24.

DeGroat WC. Integrative control of the lower urinary tract: preclinical perspective. Br J Pharmacol. 2006;147(52):S25–40.

Griffiths D. Cerebral control of bladder function. Curr Urol Rep. 2004;5:348–52.
Bannowsky A, Juenemann KP. Innervation and function of the female urinary bladder and urethra. EAU Updat Ser. 2003;1:120–7.
Bradley WE. Physiology of the urinary bladder. In: Walsh PC, et al., editors. Campbell's urology. Philadelphia: WB Saunders; 1986.

Voiding Dynamic in Women

Kira S, Mitsui T, Kobayashi H, Haneda Y, Sawada N, Takeda M. Detrusor pressures in urodynamic studies during voiding in women. Int Urogynecol J. 2017;28:783–7.
Onyishi SE, Twiss CO. Pressure flow studies in men and women. Urol Clin North Am. 2014;41:453–67.
Bauer RM, Huebner W. Gender differences in bladder control: from babies to elderly. World J Urol. 2013;31(5):1081.
Digesu GA, Hutchings A, Salvatore S, Selvaggi L, Khullar V. Reproducibility and reliability of pressure flow parameters in women. BJOG. 2003;110:774–6.
Lemack GE, Baseman AG, Zimmern PE. Voiding dynamics in women: a comparison of pressure-flow studies between asymptomatic and incontinent women. Urology. 2002;59:42–6.
Bathia NN. Dynamics of voiding in women. Curr Opin Obstet Gynecol. 2000;12:383–6.

Chapter 2
Pretest Assessment

2.1 Background

Recently the ICS has updated the International Continence Society Good Urodynamic Practice 2002 (GUP2002) with the aim to include new evidence and information on urodynamic practice and urodynamic quality control inclusive of standard on urodynamic equipment.

It is well-known that urodynamics testing incorporates a hierarchy of different evaluations, the most comprehensive of these being the pressure flow study, to which the term "urodynamics" is often applied.

In practice, there are two principal methods of urodynamic investigation:

- **Noninvasive urodynamics**, done without the insertion of a catheter (uroflowmetry, PVR)
- **Invasive or conventional urodynamics** that involves insertion of one or more catheters into the bladder or other body cavities, i.e., rectum or vagina, (cystometry, pressure-flow study)

In addition, there are **supplementary urodynamics test** (EMG, video, urethral pressure measurements, and ambulatory urodynamics) to be used in specific clinical situations.

Not all patients need invasive urodynamics. The diagnostic process for patients with LUTD should be carefully

G. Vignoli, *Urodynamics for Urogynecologists*,
https://doi.org/10.1007/978-3-319-74005-8_2

implemented and personalized to rationally select patients for invasive UDS.

Standard urodynamic protocol should include:

- Pretest assessment
- Urodynamics testing
  - Noninvasive
  - Invasive
- Posttest patient care (invasive urodynamics only)

2.2 Pretest Assessment

According to ICS classical definition, each lower urinary tract dysfunction includes four aspects:

- Clinical condition (i.e., stress urinary incontinence, voiding dysfunction, etc.)
- Symptom (assessed by detailed history)
- Sign (verified by physical examination)
- Urodynamic observation

Note: Following the recent changing of paradigms of diagnosis and management of lower urinary tract dysfunctions, the ICI-RS has suggested that symptoms and signs that patients present with can be more precisely delineated as a syndrome. A syndrome is a complex of medical signs and symptoms that are correlated with each other. In some cases, i.e., inherited disorders like Down syndrome, signs and symptoms are so specific for the disease that the word syndrome ends up being used interchangeably for the name of the disease. In other instances, like in lower urinary tract dysfunctions, a syndrome is not specific to only one disease. Female incontinence or overactive bladder is not a disease but simply a set of signs and symptoms, i.e., a syndrome.

Each patient undergoing UDS testing, both invasive and noninvasive, should have preliminarily:

- A clinical history including a valid symptom and bother score(s) and medication list

- Relevant physical examination
- Urinalysis

This basic assessment should be implemented by a:

- Voiding diary and, optionally, a pad test

2.2.1 Clinical History

The purpose of history taking is to determine the type of urinary symptom and how it is bothersome to the patient. Several validated questionnaires can be used to evaluate bother and severity of symptoms (see Box 2.1).

Obstetric history (number of pregnancies, length of labor, size of the baby, episiotomies and tears, use of forceps during delivery) and gynecological history (menstrual cycle, menopausal status, previous surgery) should also be taken.

2.2.2 Relevant Physical Examination

An abdominal examination should always be performed before moving onto vaginal examination. The lower abdomen should be palpated or percussed to assess a distended bladder

Box 2.1: Examples of Validated Urinary Incontinence Questionnaires

- Urogenital Distress Inventory (UDI)
- Incontinence Impact Questionnaire (IIQ)
- Questionnaire for Urinary Incontinence Diagnosis (QUID)
- Incontinence Quality of Life Questionnaire (I-QoL)
- Incontinence Severity Index (ISI)
- International Consultation on Incontinence Questionnaire (ICI-Q)

suggestive for a urinary retention. Then, bimanual vaginal examination should be done to evaluate the presence of uterine, adnexal, or other pelvic masses.

2.2.2.1 Structured Vaginal Examination

The structured vaginal examination should include evaluation for prolapse, vaginal wall atrophy, pelvic muscle quality and the ability to voluntarily contract them, and urinary leakage with strain or cough. The examination is usually done in lithotomy position but may be repeated with the patient standing when it is not representative of the reported symptoms. The vaginal mucosa is first inspected for signs of atrophic vaginitis, indicating estrogen deficiency. The anterior vaginal compartment is examined next by applying a slight pressure on the posterior vaginal wall with the blade of small vaginal speculum. The positions of the urethra, bladder neck, and bladder are observed at rest and with straining to evaluate the support of these structures. With coughing and straining, the urethra should be observed for urine leakage (positive stress test) and whether that leakage occurs with urethral hypermobility. A Q-tip test may be performed to assess the degree of urethral hypermobility. The central vaginal compartment is examined next. This is best accomplished by gently retracting both the anterior and posterior vaginal walls. The uterus and cervix are evaluated at rest and during straining to determine prolapse. If the patient had a hysterectomy, the vaginal cuff should be assessed for an enterocele. Finally the posterior vaginal compartment is examined by retracting the anterior vaginal wall with the speculum blade. Sometimes it may be difficult to distinguish between a high rectocele and an enterocele. With simultaneous vaginal and rectal examination, one may palpate the enterocele sliding above the anterior rectal wall. For detailed description of POPQ assessment, see Chap. 8.

Physical examination should also include routinely:

- Focused neuro-urological assessment
- Pelvic floor muscle assessment

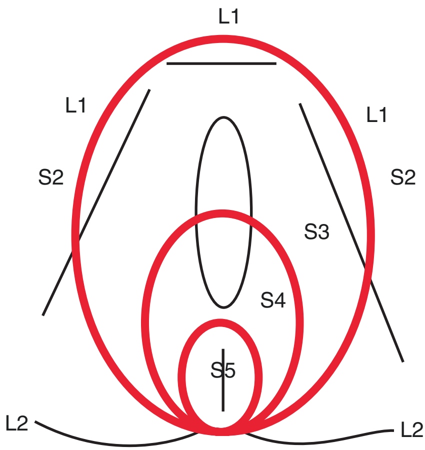


FIGURE 2.1 Focused neuro-urological examination: perineal dermatomes

2.2.2.2 Focused Neuro-urological Assessment

Observation of the patient's gait, assessment of elementary lower limb reflexes, and evaluation of sensation in different perineal dermatomes (S2–S5) (Fig. 2.1) and volitional control of the anal sphincter are important in the identification of neuro-urological abnormalities. In particular, special attention should be paid to "saddle sensation," tone, and volitional control of the anal sphincter and some reflexes that are specific for the sacral function including:

- Anal reflex: pricking the perianal skin, the anal sphincter contracts reflexly.
- Bulbocavernosus reflex: pricking the skin along the labia leads to a contraction of the anal sphincter (Fig. 2.2).

In patients found to have a neuro-urological abnormality, a complete neurological evaluation should be planned.

2.2.2.3 Pelvic Floor Muscle Assessment

Pelvic floor muscles play an important role in continence and support of the pelvic organs. Assessment of pelvic floor muscle strength can be useful in deciding nonsurgical treatment.

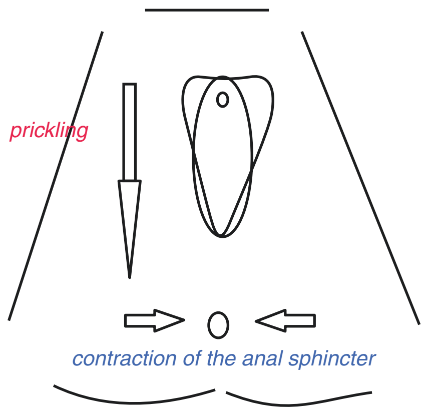


FIGURE 2.2 Focused neuro-urological examination: bulbocavernosus reflex

PFM evaluation can easily be performed by instructing the patient to contract their PFM and vaginally palpating the effect. Several components of PFM contractility can be assessed including power (strength); endurance, i.e., the length of time that a maximal voluntary contraction can be sustained (usually 10 s); the number of maximal voluntary contractions necessary to overload the muscle (usually 10); and the number of fast contractions that the subject is able to do after a short rest and the presence of co-contractions.

Power is mostly measured using Oxford grading scale (see Table 2.1).

Overall, it is difficult to evaluate and compare PFM functioning, because there are many different methods and scales. Over the past 20 years, three pelvic floor muscle assessments have been used most commonly: the Laycock PERFECT scale with a six-point scale to score muscle strength and endurance, the number of repetitions, and fast contractions; the Brink score to assess muscle strength, urethral lift, and muscle endurance, all with four-point scales; and the Devreese assessment scale, with more PFMF items, including co-contraction of the transverse abdominal muscles. In 2005 the ICS standardized the terminology relative to pelvic floor muscle functioning. In particular they redefined the muscle strength as absent, weak, normal, or strong.

Table 2.1 Oxford grading scale of muscle contraction

Grading	Muscle response
0	Nil
1	Weak
2	Flicker
3	Moderate
4	Good
5	Strong

In current clinical practice, the PFM are generally only tested for strength and endurance, but it is not yet clear whether these two functions cover total PFMF as proposed by the ICS terminology.

Note: When possible the above PFMF should be documented for each side of pelvic floor in order to differentiate unilateral defects and asymmetry.

2.2.3 Urinalysis

Before UDS testing it is considered standard to perform a urinalysis by either using a dipstick test or examining the sediment of urine. If a dipstick test is chosen, it is recommended that the strip that includes fields for hematuria, glucose, leukocyte esterase, and nitrite tests should be used. Dipstick is not as accurate as urine culture, being specific for infection but not sensitive.

Note 1: Historically urine was considered to be sterile, and only recently, utilizing new molecular technique like PCR and 16S ribosomal RNA sequencing, it has been appreciated that the bladder contains its own microbiome (saprophytic bacterial communities). Its role in different urological entities is still under investigation.

Note 2: The clinical relevance of asymptomatic bacteriuria (without pyuria) and pyuria (without bacteriuria) in the elderly is controversial.

2.2.4 Voiding Diary

Voiding diary is a specific urodynamic investigation recording fluid intake and urine output per 24-h period. Voiding diary is an important and objective tool that can be used to assess LUTD in patients seeking the diagnosis and treatment of their bladder or voiding conditions.

The recording of micturition events can be done in three main forms:

- *Micturition time chart* that records only the timing of voids, day and night, for at least 24 h.
- *Frequency volume chart (FVC)* that records the time of each micturition and the voided volume for at least 24 h.
- *Bladder diary* is the most complete form of recording and includes fluid intake, incontinence episodes, pad usage, the degree of incontinence as well as a record of episodes of urgency and sensation, and activities performed during or immediately preceding the involuntary loss of urine. (Fig. 2.3)

Bladder diary is the most suitable for a comprehensive evaluation of urinary symptoms. However, documentation

24 Hour Bladder Diary			Date 12/03/14		
Time	Drinks		Urine		Pads
	Amount (ml)	Type	Amount (ml)	Bladder Sensation	
6am WOKE			400	2	
7 am	100	Water			
8 am			150	2	
9 am					
10 am	Cup	Tea	LEAK	3	✓
11 am					
Midday					

FIGURE 2.3 Sample of ICS paper voiding diary

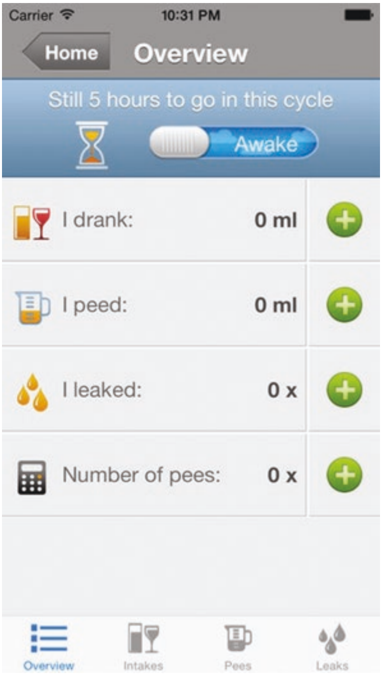


FIGURE 2.4 Screenshot of digital voiding diary, *courtesy of Synappz BV*

of the frequency with each voided volume for at least 24 h can already be extremely helpful in the initial assessment of the patient. To facilitate patient compliance, several apps for the iPhone and iPod are available on the market to create a quick and reliable digital voiding diary (Fig. 2.4).

Note: Two to three days of recording generally provide more useful clinical data.

The important parameters documented by a bladder diary include:

- Urinary frequency during the day and night
- For practical purposes, normal voiding frequency varies between five and eight times per 24 h in women and increases with age (8.2 in the sixth decade), while voiding once a night is normal for women over 75.

Note: The ICS definition of daytime frequency is when the patient considers that she is voiding too often. Therefore, no absolute number can be applied to define a cutoff value for voiding frequency.

- Functional bladder capacity, i.e., the largest volume recorded
- Typical voided volume, i.e., the most frequently observed bladder volume which may reflect the usual voided volume or the most frequent bladder sensation with respect to voiding desire
- Urgency or urgency incontinence episodes, i.e., discomfort voiding sensation felt by the patient which usually cannot be deferred
- The volume of water drunk
- Daily and nocturnal urine output (consisting of all voids during sleep and the early morning void)

Polyuria is defined as a production of more than 2.8 L of urine in 24 h. Polyuria can be due to an excessive fluid intake (polydipsia) or owing to diabetes mellitus and diabetes insipidus.

Nocturnal polyuria is defined by a nocturnal voided volume greater than 20–30%, depending on age, of total daily urine output. Nocturnal polyuria is a heterogeneous condition in which water diuresis, solute diuresis, or a combination of both is the underlying cause. Water diuresis is represented by low osmolarity at night and is due to a low ADH excretion, while nocturnal sodium diuresis is mostly due to cardiac decompensation. Accordingly, treatment will be with vasopressin at night in the first case and with diuretics during the day in the second case.

2.2.5 Pad Test

Pad test (Fig. 2.5) is complementary to voiding diary. The test assesses the degree of incontinence in a semi-objective manner. Pad weight gain in non-menstruating women can be attributed mainly to urine and in small part to perspiration and vaginal discharges. There are two types of pad test:

FIGURE 2.5 Pad test apparatus

- The short-term (1 h) pad test
- The long-term (24 h) pad test

Incontinence is diagnosed if pad weight is more than 1 g/h on the short-term pad test and more than 8 g/24 h on the long-term pad test. However, there are conflicting data concerning the correlation between severity of incontinence and increase in pad weight.

Suggested Reading

Overviews

Rosier PFWM, Schaefer W, Lose G, Goldman HB, Guralnick M, Eustice S, et al. International continence society good urodynamic practices and terms 2016: urodynamics, uroflowmetry, cystometry, and pressure-flow study. Neurourol Urodyn. 2017;36:1243–60.

Medina C, Costantini E, Petri E, Mourad S, Singla A, Rodríguez-Colorado S, et al. Evaluation and surgery for stress urinary incontinence: a FIGO working group report. Neurourol Urodyn. 2017;36:518–28.

Rosier P, Giarenis I, Valentini F, Wein A, Cardozo L. Do patients with symptoms and signs of lower urinary tract dysfunction need a urodynamic diagnosis? Neurourol Urodyn. 2014;33:581–6.

Winters JC, Dmochowski RR, Goldman HB, Herndon CD, Kobashi KC, Kraus SR, et al. Urodynamic studies in adults: AUA/SUFU guideline. J Urol. 2012;188:2464–72.

Lenherr SM, Clemens JQ. Urodynamics with a focus on appropriate indications. Urol Clin North Am. 2013;40:545–57.

Haylen BT, de Ridder D, Freeman RM, Swift SE, Berghmans B, Lee J, et al. An International Urogynecological Association (IUGA)/International Continence Society (ICS) joint report on the terminology for female pelvic floor dysfunction. Neurourol Urodyn. 2010;29:4–20.

Questionnaires

Shumaker SA, Wyman JF, Uebersax JS, McClish D, Fantl JA. Health-related quality of life measures for women with urinary incontinence: the Incontinence Impact Questionnaire and the Urogenital Distress Inventory. Continence Program in Women (CPW) Research Group. Qual Life Res. 1994;3:291–306.

Uebersax JS, Wyman JF, Shumaker SA, McClish DK, Fantl JA. Short forms to assess life quality and symptom distress for urinary incontinence in women: the Incontinence Impact Questionnaire and the Urogenital Distress Inventory. Continence Program for Women Research Group. Neurourol Urodyn. 1995;14:131–9.

Abrams P, Avery K, Gardener N, Donovan J, ICIQ Advisory Board. The international consultation on incontinence modular questionnaire: www.iciq.net. J Urol. 2006;175:1063–6.

Coyne KS, Thompson CL, Lai JS, Sexton CC. An overactive bladder symptom and health-related quality of life short-form: validation of the OAB-q SF. Neurourol Urodyn. 2015;34:255–63.

Bradley CS, Rahn DD, Nygaard IE, Barber MD, Nager CW, Kenton KS, et al. The questionnaire for urinary incontinence diagnosis (QUID): validity and responsiveness to change in women under-

going non-surgical therapies for treatment of stress predominant urinary incontinence. Neurourol Urodyn. 2010;29:727–34.

Brown JS, Grady D, Ouslander JG, Herzog AR, Varner RE, Posner SF. Prevalence of urinary incontinence and associated risk factors in postmenopausal women. Heart & Estrogen/Progestin Replacement Study (HERS) Research Group. Obstet Gynecol. 1999;94:66–70.

Sandvik H, Seim A, Vanvik A, Hunskaar S. A severity index for epidemiological surveys of female urinary incontinence: comparison with 48-hour pad-weighing tests. Neurourol Urodyn. 2000;19:137–45.

Avery K, Donovan J, Peters TJ, Shaw C, Gotoh M, Abrams P. ICIQ: a brief and robust measure for evaluating the symptoms and impact of urinary incontinence. Neurourol Urodyn. 2004;23:322–30.

Physical Examination

Panicker JN, Fowler CJ, Kessler TM. Lower urinary tract dysfunction in the neurological patient: clinical assessment and management. Lancet Neurol. 2015;14:720–32.

Al Afraa T, Mahfouz W, Campeau L, Corcos J. Normal lower urinary tract assessment in women: I. Uroflowmetry and post-void residual, pad tests, and bladder diaries. Int Urogynecol J. 2012;23:681–5.

Newman D, Laycock J. Clinical evaluation of pelvic floor muscles. In: Baessler K, Burgio K, Norton P, Schüssler B, Moore K, Stanton S, editors. Pelvic floor re-education. Principles and practice. London: Springer; 2008. p. 91–104.

Bø K, Sherburn M. Evaluation of female pelvic floor muscle function and strength. Phys Ther. 2005;85:269–82.

Messelink B, Benson T, Berghmans B, Bø K, Corcos J, Fowler C. Standardization of terminology of pelvic floor muscle function and dysfunction: report from the pelvic floor clinical assessment group of the International Continence Society. Neurourol Urodyn. 2005;24:374–80.

Laycock J, Jerwood D. The pelvic floor muscles assessment. The PERFECT scheme. Physiotherapy. 2001;87:631–42.

Brink CA, Sampselle CM, Wells TJ, Diokno AC, Gl G. A digital test for pelvic muscle strength in older women with urinary incontinence. Nurs Res. 1989;38:196–9.

Devreese A, Staes F, Weerdt W, Feys H, Van Assche A, Penninckx F, et al. Clinical evaluation of pelvic floor muscle function in continent and incontinent women. Neurourol Urodyn. 2004;23:190–7.
Nitti V, Ficazzola M. Voiding dysfunction: diagnostic evaluation. In: Appel R, editor. Voiding dysfunction: diagnosis and treatment. Humana Press; 2000.

Voiding Diaries

Bright E, Drake MJ, Abrams P. Urinary diaries: evidence for the development and validation of diary content, format, and duration. Neurourol Urodyn. 2011;30:348–52.
Van Kerrebroeck P, Andersson KE. Terminology, epidemiology, etiology, and pathophysiology of nocturia. Neurourol Urodyn. 2014;33(Suppl 1):S2–5.
Goessaert AS, Krott L, Hoebeke P, Vande Walle J, Everaert K. Diagnosing the pathophysiologic mechanisms of nocturnal polyuria. Eur Urol. 2015;67:283–8.

UTI

Mody L, Juthani-Mehta M. Urinary tract infections in older women: a clinical review. JAMA. 2014;26(311):844–54.
Buchsbaum GM, Albushies DT, Guzick DS. Utility of urine reagent strip in screening women with incontinence for urinary tract infection. Int Urogynecol J Pelvic Floor Dysfunct. 2004;15:391–3.
Yu Y, Sikorski P, Bowman-Gholston C, Cacciabeve N, Nelson KE, Pieper R. Diagnosing inflammation and infection in the urinary system via proteomics. J Transl Med. 2015;13:111.

Pad Test

Krhut J, Zachova RL, Smith P, Rosier PF, Valanský L, Martan A, et al. Pad weight testing in the evaluation of urinary incontinence. Neurourol Urodyn. 2014;33:507–10.
Costantini E, Lazzeri M, Bini V. Sensitivity and specificity of one-hour pad test as a predictive value for female urinary incontinence. Urol Int. 2008;81:153–9.

Chapter 3
Noninvasive Urodynamics

3.1 Background

Noninvasive urodynamics in female consists of two tests: uroflowmetry and postvoid residual urine (PVR) assessment. From PVR and voided volume, a voided percentage (Void%) can be calculated. While these tests are useful tools for screening, they have evident limits since subsequent invasive tests are necessary to confirm the diagnosis and refine the findings. Owing to their limitations, such tests must always be interpreted by an experienced urogynecologist with knowledge of patient's complaints and symptoms.

3.1.1 Uroflowmetry

Uroflowmetry is the analysis of urine flow rate over the time. The technique is a simple way to measure bladder emptying and is the first-line screening test for most patients with LUTS. By itself, a uroflow is seldom able to determine the cause of voiding dysfunction; however, in conjunction with a careful history and physical examination, it can provide valuable information and select the patients for further invasive urodynamic investigation.

G. Vignoli, *Urodynamics for Urogynecologists*,
https://doi.org/10.1007/978-3-319-74005-8_3

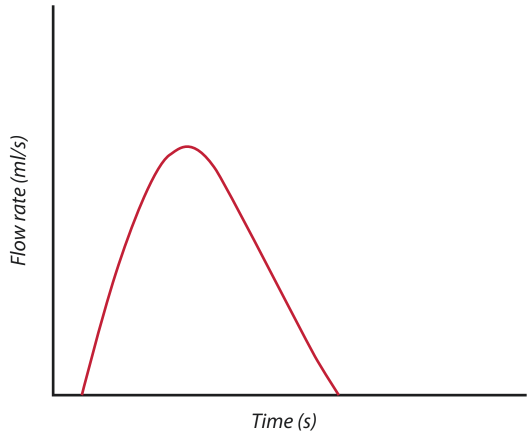


FIGURE 3.1 Uroflowmetry curve

Uroflow is measured by a collecting electronic equipment (uroflowmeter) which provides a graphic expression of the flow rate as a function of time (Fig. 3.1).

Since uroflowmetry results are sensitive to patient cooperation and emotion, all free uroflowmetry studies should be performed in a completely private dedicated room. Females usually void seated on the commode except patients with voiding dysfunction which could prefer the standing position. When flow rate and voided volume are unexpectedly low and conversely PVR is larger than expected basing on history, emotional problems should be taken into account, and the test should be repeated.

3.1.1.1 Interpretation of Uroflowmetry

Several parameters from the trace are automatically calculated and printed out together with the trace.

Note: In automated calculations, it is important that the examiner verifies the presence of artifacts such as an accidental knock to the flowmeter and, in case, checks the parameters of the curve manually (Fig. 3.2).

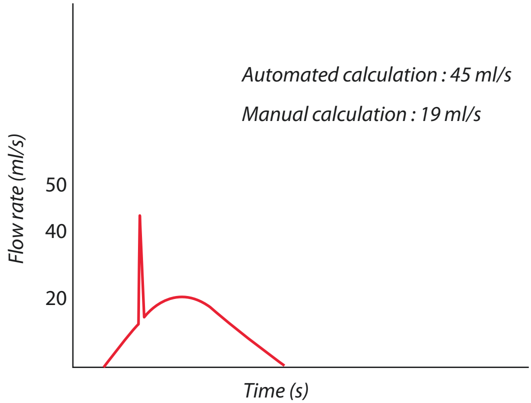


FIGURE 3.2 Common uroflowmetry artifact: accidental knock to the equipment

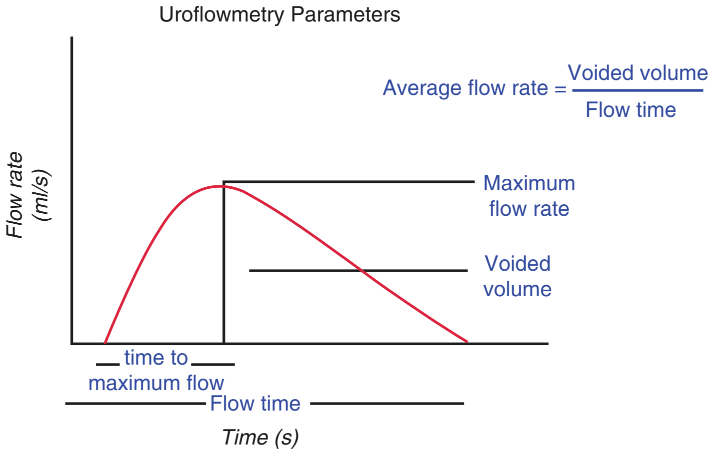


FIGURE 3.3 Parameters of uroflowmetry curve

Common parameters determined by uroflowmetry include (Fig. 3.3):

- *Maximum flow rate (Qmax)*, that is, the maximum measured value of the flow curve.
- *Flow time*, that is, the time over which a measurable flow occurs. Flow time is easily measured if the flow is continuous.

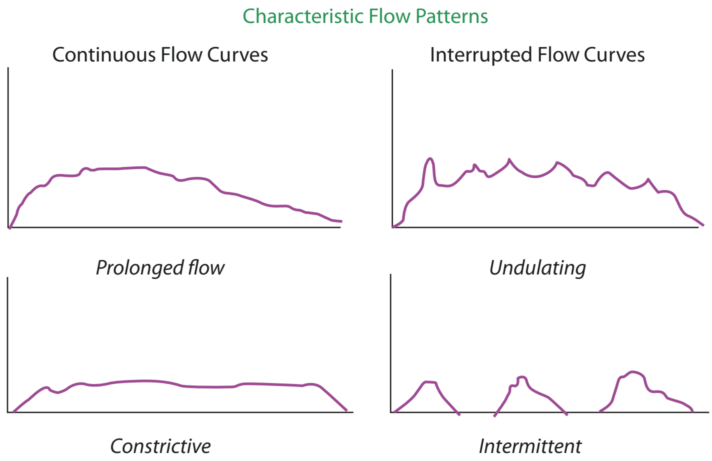


FIGURE 3.4 Patterns of uroflowmetry curve

When urine flow is intermittent, the time intervals between flow episodes are not considered. Similarly, when there is a lengthy terminal dribbling, it may be noted but not included in the flow time.

- *Average flow rate*, that is, the volume voided divided by the flow time.
- *Time to maximum flow*, that is, the elapsed time from onset of flow to maximum flow rate.
- *Morphology of the curve*. Flow curve shape is complementary to flow rate in the assessment of voiding. Despite some disparities in the definition of the morphology of flow curve, several patterns of flow curves can be recognized (Fig. 3.4):
  - Normal flow curve is continuous and has a bell shape with steep slope and short flow time.
  - Long flow, a long and constant flow rate during the entire micturition.
  - Constrictive, flat plateau of low flow.
  - Undulating with multiple peaks: a long continuous curve with multiple peaks, each representing 20% or more than Qmax frequently seen in dysfunctional voiding.

– Interrupted curve. Discontinuous curve characterized by repetitive flow peaks reaching zero in between, which can be due to abdominal straining or detrusor sphincter dyssynergia.

A long flow curve may be due either to urethral obstruction or to poor detrusor contraction. A pressure-flow study is necessary to clarify the underlying mechanism. A constrictive curve may be seen in urethral stricture (i.e., after placement of obstructive mid-urethral sling).

In neurologically normal patients, undulating flow curve is most often a result of anxiety or dysfunctional voiding. Intermittent flow in neurologic patients may be a consequence of detrusor sphincter dyssynergia in suprasacral lesions or abdominal straining in sacral and sub-sacral lesions. Again a pressure-flow study is mandatory to define the exact mechanism.

Qmax in women ranges from 23 to 33 mL/s.

Unlike men, in women the relation between Qmax and age is unclear. The same concept applies to pre-/postmenopausal status. Some studies report a negative relation probably linked to the development of detrusor underactivity with aging, while other studies do not indicate any significant connection.

Voiding time in women is lower than in men due to the shortness of the urethra and is about 20 s. Flow rate is closely related to voided volume. Voided volumes lower than 150 mL are considered unreliable, and the test should be repeated asking the patient to drink water until she experiences a strong but not uncomfortable desire to void. On the other hand, a voided volume greater than 700 mL is usually associated with a decline of Qmax.

To overcome the problem of volume dependency, Haylen constructed Liverpool nomograms based on maximum and average flow rates of healthy asymptomatic volunteers over a wide range of voided volumes (15–600 mL). Nomogram is in centile form. Flow rates below the fifth centile may be generally regarded as abnormal; abnormality can be suspected in flow curves between fifth and tenth centiles (Fig. 3.5).

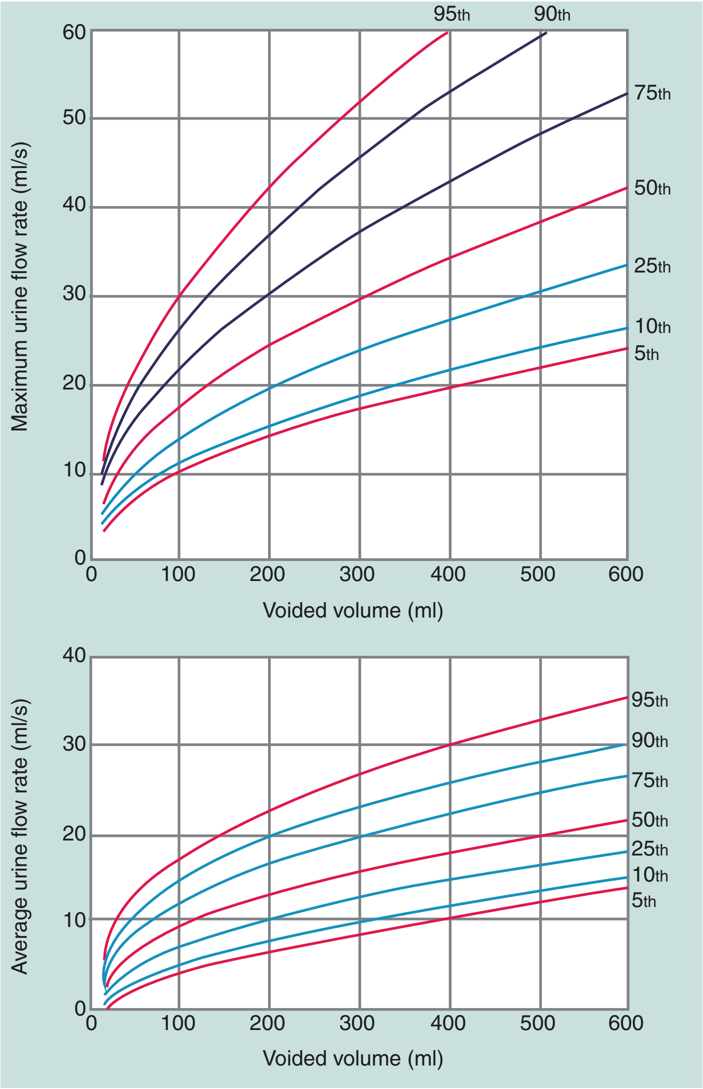


FIGURE 3.5 The Liverpool nomogram for maximum and average flow for women

3.1.2 Postvoid Residual (PVR) Assessment

The postvoid residual volume (PVR) is defined as the volume of urine remaining in the bladder immediately following voiding. It provides information on the ability of the bladder to empty as well as its functional capacity (voided volume plus PVR). Normal lower urinary tract function is usually associated with a negligible PVR (<50 mL); however, there is no agreement as to what an "abnormal" or clinically significant value is. Thus, elevated PVR is somewhat arbitrary. This being the case, most would agree that a PVR of greater than 100 mL is elevated, although perhaps not significant. Elevated PVR may be an indication of detrusor hypocontractility or bladder outlet obstruction and may require further evaluation depending on the patient and the symptoms complained. PVR can be measured directly by urethral catheterization or, better, determined noninvasively by ultrasonography. Portable bladder scanners based on ultrasound technology are currently available to determine bladder volume (Fig. 3.6).

3.1.3 Voided Percentage (Void%)

Void% is the numerical description of the voiding efficacy or efficiency which is the proportion of bladder content emptied. Void% can be calculated by the formula: [(volume voided/volume voided + PVR) × 100].

FIGURE 3.6 The bladder scanner, *courtesy of Echo-Son*

Example: If a patient voids 300 mL leaving a postvoid residual volume of 100 mL (total bladder capacity, 400 mL), then she has a Void% of 75.

Voiding efficiency may be a relevant parameter in addition to Qmax and PVR for the follow-up of treated patients.

Key Points

- Uroflowmetry helps to identify patients with voiding problems. However, by itself, the procedure cannot distinguish true obstruction from poor bladder contractility. Thus, the test's utility lies in its ability to identify patients who need further urodynamic studies.
- Measurement of postvoid residual urine volume (PVR) is also useful in assessing voiding dysfunction.
- PVR measurement can be performed by sterile catheterization or by bladder ultrasound. Portable bladder scanners are convenient, noninvasive, accurate, and cost-effective and carry no risk of urethral trauma or urinary tract infection.
- However, there is no agreement as to what is an "abnormal" PVR: a PVR lower than 50 mL is negligible, while a PVR greater than 100 mL is probably elevated, although perhaps not significant.
- Void% may be an additional parameter in the evaluation of voiding process.

Suggested Reading

Uroflowmetry

Sorel M, Reitsma H, Rosier P. Uroflowmetry in healthy women: a systematic review. Neurourol Urodyn. 2017;36:953–9.

Rane A, Iyer J. Posture and micturition: does it really matter how a woman sits on the toilet? Int Urogynecol J. 2014;25:1015–21.

Al Afraa T, Mahfouz W, Campeau L, Corcos J. Normal lower urinary tract assessment in women: I. Uroflowmetry and post-void residual, pad tests, and bladder diaries. Int Urogynecol J. 2012;23:681–5.

Haylen BT, de Ridder D, Freeman RM, Swift SE, Berghmans B, Lee J, et al. An International Urogynecological Association (IUGA)/International Continence Society (ICS) joint report on the terminology for female pelvic floor dysfunction. Neurourol Urodyn. 2010;29:4.

Yang KN, Chen SC, Chen SY, Chang CH, Wu HC, Chou EC. Female voiding postures and their effects on micturition. Int Urogynecol J. 2010;21:1371.

Barapatre Y, Agarwal MM, Singh SK, Sharma SK, Mavuduru R, Mete UK, et al. Uroflowmetry in healthy women: development and validation of flow-volume and corrected flow-age nomograms. Neurourol Urodyn. 2009;28:1003.

Kumar V, Dhabalia JV, Nelivigi GG, Punia MS, Suryavanshi M. Age, gender, and voided volume dependency of peak urinary flow rate and uroflowmetry nomogram in the Indian population. Indian J Urol. 2009;25:46.

Haylen BT, Yang V, Logan V. Uroflowmetry its current clinical utility for women. Int Urogynecol J Pelvic Floor Dysfunct. 2008;19:899–903.

Pauwels E, De Laet K, De Wachter S, Wyndaele JJ. Healthy, middle-aged, history-free, continent women—do they strain to void? J Urol. 2006;175:1403.

Haylen BT, Law MG, Frazer M, Schulz S. Urine flow rates and residual urine volumes in urogynecology patients. Int Urogynecol J Pelvic Floor Dysfunct. 1999;10:378–83.

Wyndaele JJ. Normality in urodynamics studied in healthy adults. J Urol. 1999;161:899.

Abrams P. Bladder outlet obstruction index, bladder contractility index and bladder voiding efficiency: three simple indices to define bladder voiding function. BJU Int. 1999;84:14–5.

Haylen BT, Ashby D, Sutherst JR, Frazer MI, West CR. Maximum and average urine flow rates in normal male and female populations—the Liverpool nomograms. Br J Urol. 1989;64:30–8.

Fantl JA, Smith PJ, Schneider V, Hurt WG, Dunn LJ. Fluid weight uroflowmetry in women. Am J Obstet Gynecol. 1983;145:1017.

Post-Void Residual Urine

Asimakopoulos A, De Nunzio C, Kocjancic E, Tubaro A, Rosier P, Finazzi-Agrò E. Measurement of post-void residual urine. Neurourol Urodyn. 2016;35:55–7.

Goode PS, Locher JL, Bryant RL. Measurement of post-void residual urine with portable transabdominal bladder ultrasound scanner and urethral catheterization. Int Urogynecol J Pelvic Floor Dysfunct. 2000;11:296–300.

Chapter 4
How to Perform Conventional Urodynamic Investigation: The "Good Urodynamic Practice"

4.1 Background

Invasive or conventional urodynamic testing includes:

- Transurethral cystometry for the assessment of bladder filling
- Pressure/flow study for the assessment of voiding phase

The performance, the results, and the interpretation of urodynamic testing are dependent on both the urodynamicist involved and the equipment being used. Rapid development of computer-based technology has led to sophisticated urodynamic equipment (Fig. 4.1).

Therefore, it is of greatest importance that the clinician performing the test is familiar with the urodynamic software and all the equipment that is used during the study. Proper calibration and standardization of equipment, together with the expertise of the clinician, who should understand the patients' symptoms and decide on more appropriate test, are of paramount importance for "a good urodynamic practice."

Note: UDS may be inconvenient for some patients, resulting in poor cooperation, which may alter the accuracy of the

G. Vignoli, *Urodynamics for Urogynecologists*,
https://doi.org/10.1007/978-3-319-74005-8_4

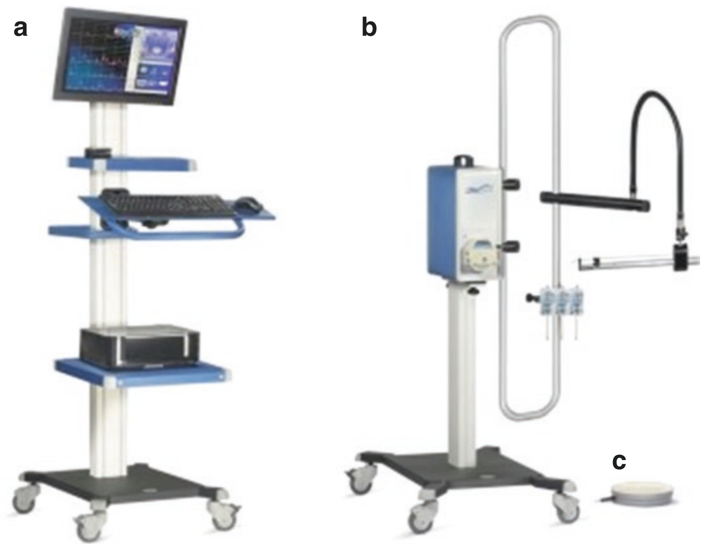


FIGURE 4.1 Equipment for conventional urodynamic testing including (**a**) pressure/flow workstation with superimposed X-ray image, (**b**) urethral profilometer, and (**c**) uroflowmeter, *courtesy HC Italia*

results. Conflicting evidence exists about which precise information is helpful to give to patients before urodynamic testing to reduce distress. Often information leaflets about urodynamic investigations are too difficult for patients to understand. A good information with clear and unambiguous wording may increase patient acceptance and confidence reducing the stress of investigation.

4.2 Setup of the Equipment

Three types of pressures are recorded during standard urodynamic test (Fig. 4.2):

- *Vesical* is the pressure recorded within the bladder and, as such, is the only absolute pressure recorded.
- *Abdominal* is the pressure surrounding the bladder and indicates both passive forces created when the abdominal

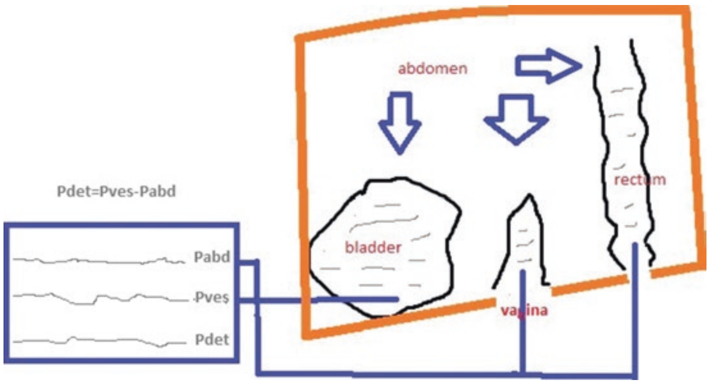


FIGURE 4.2 Pressures measured during conventional urodynamic testing

structures lie on the bladder during patient's position changes (from supine to sitting or upright position) and active forces created by physical exertion like coughing or sneezing. Measuring Pabd helps the examiner differentiate the effects of abdominal from detrusor forces. Pabd can be measured in various locations, including the rectal vault (most commonly), posterior vaginal vault, and abdominal stoma in patient with urinary or fecal diversion. Regardless of where it is measured, Pabd is a relative pressure; in other words, it provides a reasonable estimate of abdominal forces, but it is not possible to directly measure Pabd in the clinical setting. Furthermore it is vulnerable to physiological artifacts like bowel contractions when measured in the rectal vault or movements of fluid-filled balloon when measured in the posterior vaginal vault of a woman with significant pelvic organ prolapse.

- *Detrusor* is the pressure created by the tone of the smooth muscle within the bladder wall and passive forces exerted by the viscoelastic properties of the bladder wall. It is calculated by subtracting Pabd from Pves (Pdet = Pves – Pabd). Since Pdet is derived by subtracting a relative pressure (Pabd) from an absolute pressure (Pves), it is also a relative pressure and as such well subject to physiological artifacts.

4.3 Transducers

Pressure measurement can be obtained using different types of transducers.

4.3.1 Water-Filled (External) Transducers (Fig. 4.3)

These are the most common transducers and are located between the patient and the urodynamic equipment. Transmission lines extend from the catheters placed in the patient (bladder, rectum, or posterior vaginal vault) and pressure transducer (usually located on the stand), while an elec-

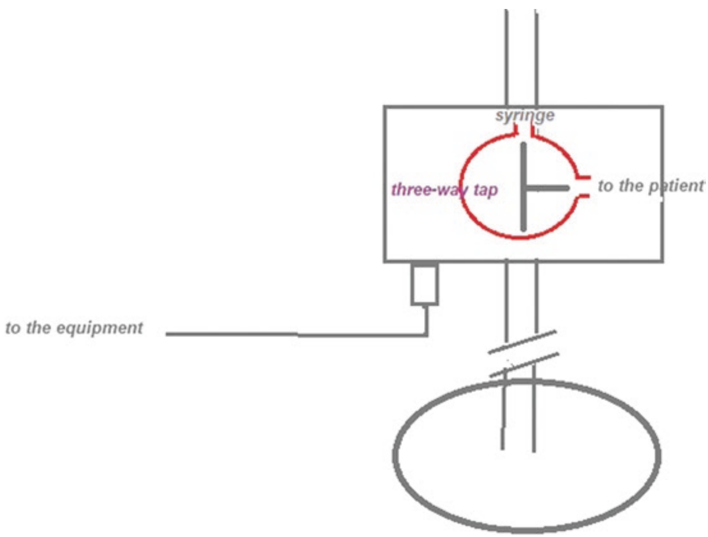


FIGURE 4.3 Schematic drawing of water-filled external transducers. Water-filled system allows the transmission of intravesical and abdominal pressures from the patient to the external pressure transducers, which are attached to the urodynamic equipment

tronic cable connects the transducer to the urodynamic equipment. Because this system depends on the transmission of pressure through fluid (water), it is crucial that there are no air bubbles in the transducer or tubing to avoid a signal dampening.

The main limitation of the water-filled system is that patient movement (i.e., provocative maneuvers to demonstrate SUI or DO) causes high-frequency artifactual signals that are transmitted to the transducer by the water column and as a result make the urodynamic traces harder to interpret. Furthermore a re-levelling of the transducers is required every time the patient moves from supine to sitting and standing positions.

4.3.2 *Microtip Transducers* (Fig. 4.4)

Strain gauges are miniaturized in order to fit the tip of a 6–8 French catheter. The external proximal end of the catheter is connected directly to the electronic recording system, thus making them simpler to set up than fluid-filled systems. In addition, a reference height does not need to be set up since unlike external fluid catheters, it is at the level of the internal transducer. Nevertheless, they need to be calibrated regularly before use.

Microtip transducers are advantageous in certain situations such as ambulatory urodynamics since they are not affected by movement artifacts, but for conventional cystometry, they are not recommended by the ICS. The main disad-

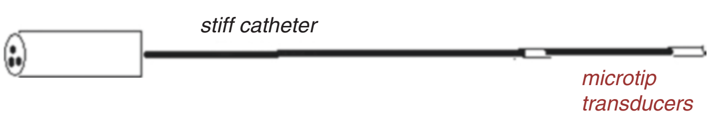


Figure 4.4 Schematic drawing of dual-sensor microtip transducer for simultaneous measurement of bladder and urethral pressure

vantage of this system is that the pressure readings are directional and rely on what the transducer is facing or what the transducer is lying against. In addition the catheters are expensive and must be handled with care.

4.3.3 *Air-Charged Catheters (T-DOC System)* (Fig. 4.5)

This is the newest type of transducer. The system consists of permanent cables with incorporated transducers and disposable catheters with a tiny balloon located on the distal end of the catheter. After the catheter is inserted into the patient and connected to the cable, the transducer is used to “charge” the catheter by injecting a micro-volume of air into the catheter balloon. This creates a closed sensitive system for accurate recording of bladder, abdominal, and urethral pressures. Air-charged catheter has gained popularity due to its simpler and quicker setup than fluid-charged systems. In addition pressure detection is not directional, and the catheters are disposable unlike the microtip catheters. However, pressures measured using air-charged catheters are not comparable with water-filled catheters and are therefore not interchange-

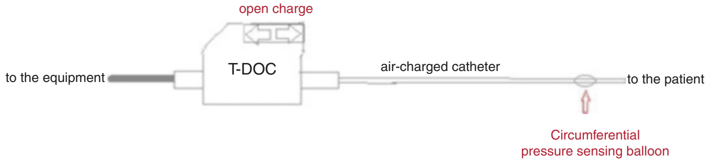


FIGURE 4.5 Schematic drawing of air-charged catheters (T-DOC system). This catheter uses a miniature, air-filled balloon placed circumferentially around a polyethylene catheter. External forces on the balloon are transmitted to the air-filled catheter lumen and communicated to an external transducer connected to the equipment

able. The air-charged catheters consistently produce higher mean pressures than the water-filled catheters. Caution, thereby, must be used when comparing urodynamic parameters using air-charged and water-filled catheters.

4.3.4 Zeroing the External Water-Filled Transducers

The pressure measurements can be “zeroed” either to the surrounding atmospheric pressure or to the internal pressure. The ICS recommend that the surrounding atmospheric pressure should be used as it facilitates standardization of the technique and comparison of data from other centers.

Zero is established by exposing the pressure transducer to the atmosphere and setting the pressure value at 0 cmH_2O.

In practice, once the bladder and rectal catheters are in place, they are connected through transmission lines to their respective transducer domes via a three-way stopcock. At the other end of the transducer dome, a 20-mL syringe filled with saline is also connected using a three-way stopcock. The syringe will be used to flush air out of the system. Zeroing the system to the atmosphere is achieved by opening the dome of the transducer to the air while the three-way stopcock connecting the patient and syringe are off and then pressing the zero button on the software system. Once this is done, the dome of the transducer is closed to the air while stopcock to syringe and patient are opened to flush saline into the tubings to eliminate air in the system. Once air bubbles are removed, the syringe stopcock is turned off, and the system, opened only to the patient, is ready for recording (Fig. 4.6).

At this point, the patient is asked to cough to check for accuracy of recorded signals.

Because a cough reflects abdominal forces acting on the bladder, Pves and Pabd tracings are expected to reflect a twin image characterized by a rapid rise and rapid descent of

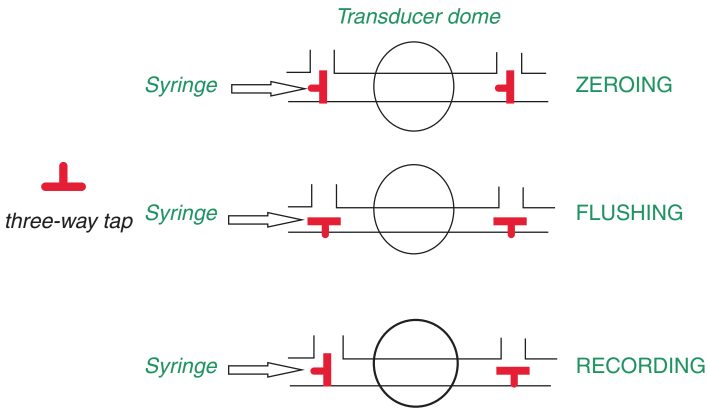


FIGURE 4.6 Three-way tap positions during setting of water-filled transducer. Various positions allow the transducer to be zeroed to atmospheric pressure, tubing to be flushed, and pressure to be recorded

pressure to baseline. In contrast, the Pdet tracing should show no response (Fig. 4.7). In fact, a small biphasic pressure spike is usually seen on the Pdet trace. The biphasic spike is an accepted artifact caused by the maximum pressure in the Pves and Pabd traces being recorded at different times.

4.3.5 Reference Height of the External Water-Filled Transducers

The reference height is the level at which the transducers must be placed so that all urodynamic pressures have the same hydrostatic component. The transducer should be positioned at the level of the pressure to be measured during zeroing and during measurement.

Since the urine into the bladder acts as a column of water, the pressure at the dome of the bladder (at the top of the column) should be lower than the pressure at the base of the bladder (at the bottom of the column of fluid). For that reason the ICS has established the superior margin of the symphysis pubis as the reference point for measuring all UDS pressures.

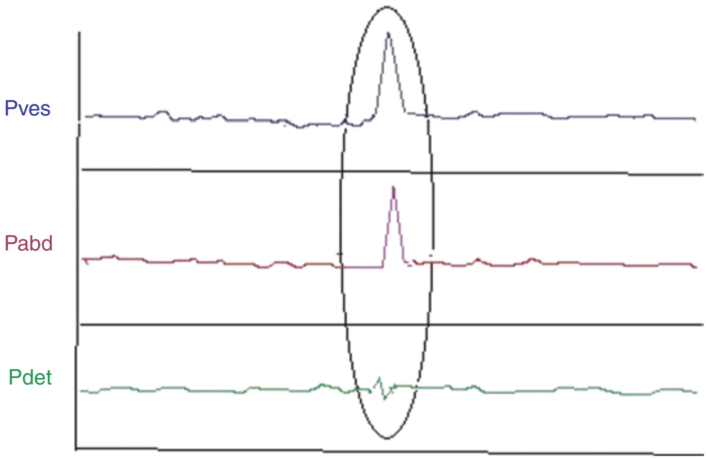


FIGURE 4.7 Cough induces near-identical spikes in Pves and Pabd, while in Pdet is seen a small biphasic spike due to the recording gap between the two previous pressures

If the patient changes the position during the test (i.e., sitting to standing), the height of the transducer should be adjusted so that it remains at the level of the bladder. Moving the patient bed during the examen (from supine to sitting and to standing position) without modifying the position of the transducer will provide inaccurate readings.

4.3.6 Microtip and Air-Filled Systems

For microtip transducers the reference height is the transducer itself; for air-filled transducers the reference height is at the position of the internal balloon. Using these systems it is therefore difficult to be certain of the position of the reference height or to ensure that the reference height of the intra-abdominal and the intravesical lines is equal. In addition changes of patient position can cause significant differences in the positions of intravesical and intra-abdominal transducers/balloons. In the supine position, the rectal line is likely to be lower than the intravesical line; when standing the rectal line

may be higher than the intravesical line. In practice, however, these differences in height are unlikely to result in a significant effect on recorded pressures, and most of the UDS instruments are equipped with an automatic zeroing system (Fig. 4.8).

Note: *Pves-Pabd* balance button in water-filled systems.

In water-filled systems, artificial adjust of the recorded pressures goes against the principles of physiological measurement, and this was described as a common mistake in Good Urodynamic Practice.

4.4 Setup of the Patient

- Preparation
  Although evidence indicates that urodynamics is generally well tolerated, embarrassment, fear of pain, and apprehension are quite common in the majority of patients. An explanatory leaflet with sufficient information about urodynamic investigation, written with clear and unambiguous words, may be particularly useful to defeat the anxiety. Furthermore, the patient should be aware that the procedure is interactive, and she should discuss her sensations during the study and whether or not her symptoms are reproduced during the study.
  Note: In any patient undergoing UDS, a conventional urinalysis or simply a urine dipstick to screen for infection should be available. When infection is present, the examen should be rescheduled after appropriate antibiotics treatment. There are no emergencies in urodynamics!
- Informed consent
  The patient should be aware of the risks of the procedure and should be informed about the possibility of a urinary tract infection, hematuria, and voiding dysfunction after the procedure (see Appendix B).
- Position
  The standard position during cystometry should be sitting position because this is reported to have a higher sensitivity for the diagnosis of abnormalities during filling phase. Also standing position can be used in patients able to do.

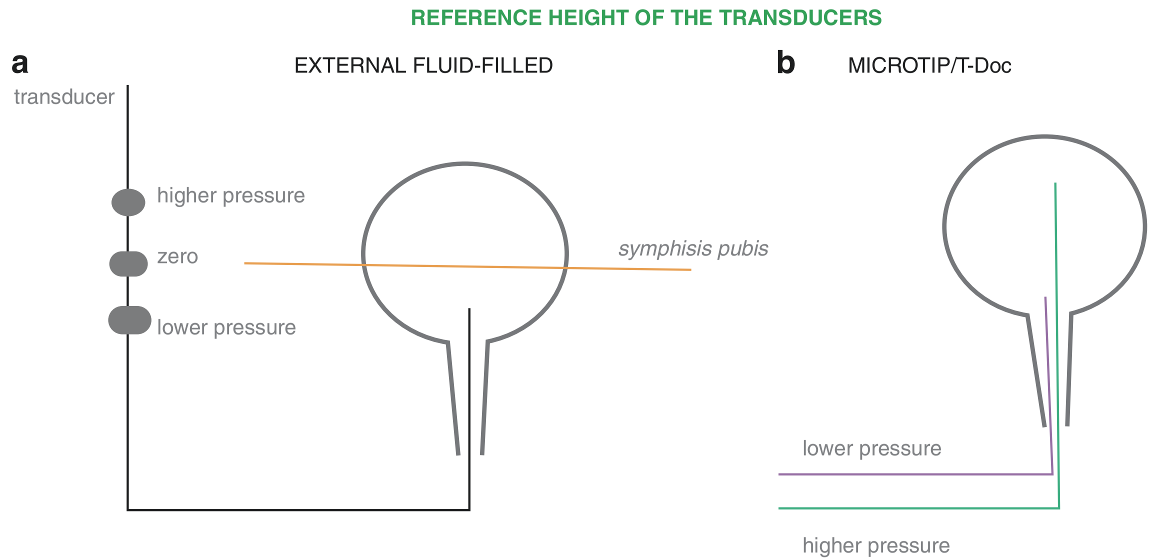


FIGURE 4.8 (**a**) With the external water-filled transducers, the reference height is the superior margin of the symphysis pubis; (**b**) for microtip and air-filled transducers, the reference height is the position of the transducer or the balloon itself

- Catheters

  Bladder: a double-lumen catheter as thin as possible (5–8 Fr) for filling and pressure recording is regarded as the gold standard (Fig. 4.9). A double-lumen catheter allows a smooth transition from storage to voiding and permits the exam to be repeated without reinsertion of a filling catheter. Ancillary dual catheter method seems disadvantageous because removal of the separate filling catheter just before micturition may interfere with lower urinary tract function and may also displace the intravesical pressure-sensing catheter.

  Rectum/vagina: abdominal pressures are measured with a balloon catheter (Fig. 4.10) inserted into the rectum for 5–10 cm. The balloon should be filled with a small amount of fluid (10–20% of its capacity) to prevent obstruction of the catheter by the rectal content. Overfilling is a common mistake which can cause misleading measurements. To prevent overfilling the balloon can be punctured, but pressure can also be recorded through an open fluid-filled tube without the balloon. As alternative to the rectum, the catheter can be positioned in the vaginal vault or stoma when the rectum is closed, although the pressures recorded, in the latter case, seem less reliable.

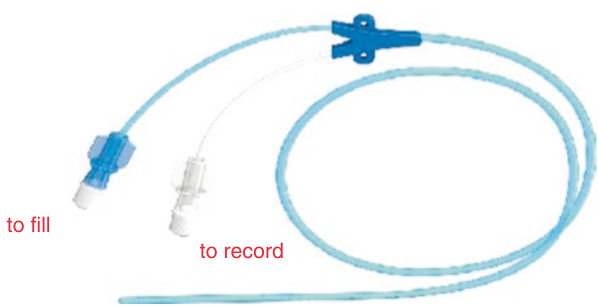


FIGURE 4.9 Double-lumen catheter (5–8 Fr) for filling and recording bladder pressure

FIGURE 4.10 Rectal/vaginal balloon catheter

- EMG electrodes
  Electromyography of pelvic floor muscles is a supplementary urodynamic test and is usually indicated in neurological patients and in dysfunctional voiding. Two types of electrodes are usually employed: surface and needle/wire (see Chap. 6).
  Note: Bladder and rectal catheters as well as EMG electrodes are positioned with the patient in supine position and should be securely fixed with adhesive tape (Fig. 4.11).
- Filling medium
  Saline solution at room temperature is the commonly used fluid for bladder filling. When videourodynamics is performed, a contrast solution is added.
  The rate of filling is usually 50 mL/min in non-neurogenic patient and 20 mL/min in neurogenic patient more prone to develop detrusor overactivity with a high filling rate.
- Display of signals
  All measured (Pabd and Pves) and derived (Pdet) signals are displayed according to the ICS *standards (Good urodynamic practice, 2002)* (Fig. 4.12):
  - *Vesical pressure*
  - *Abdominal pressure*
  - *Detrusor pressure*
  - *Flow*

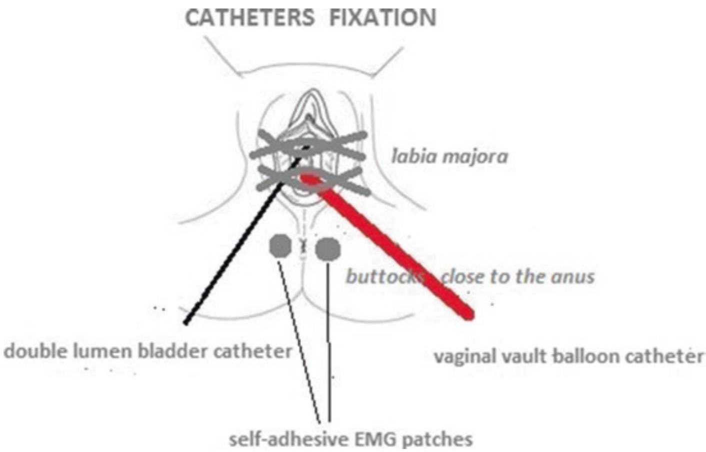


FIGURE 4.11 Catheters and EMG electrodes should be securely fixed with the patient in supine position

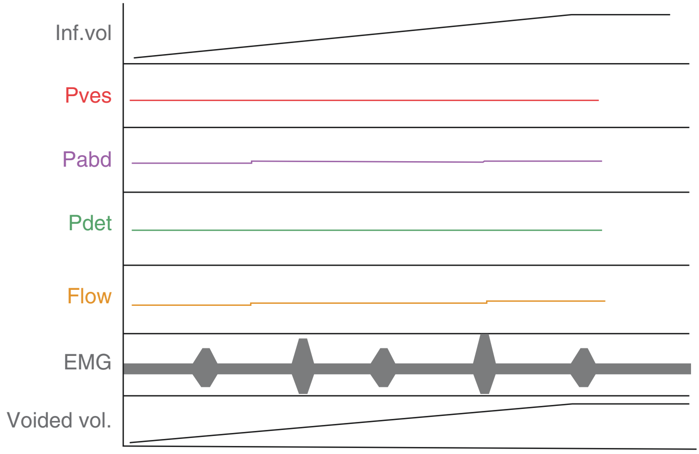


FIGURE 4.12 Display of tracings: in color, the ICS recommended sequence of basic tracings (Pves, Pabd, Pdet, Flow) and in gray, the sequence of optional tracings (infused volume, EMG, voided volume)

Filling volume, electromyography, and voided volume are displayed in additional curves.

- Resting pressures

  The initial resting pressures, if transducers are properly zeroed and positioned, are 15–40 cmH_2O (sitting) or 30–50 cmH_2O (standing), both for the vesical and intrarectal pressures. By consequence the subtracted detrusor pressures are around zero. In the supine position, the vesical pressure will be 5–20 cmH_2O and the intrarectal pressure usually somewhat higher as a consequence of the position. Then small differences in detrusor pressures (<+or – 10 cmH_2O) can be considered to be a result from differences in catheter tip position of bladder and rectal catheters and are therefore acceptable (Fig. 4.13). If the pressure ranges are outside the acceptable range, two possibilities have to be considered:

  – When vesical and abdominal pressures are similar but outside the expected range, the height of the transducer should be checked.

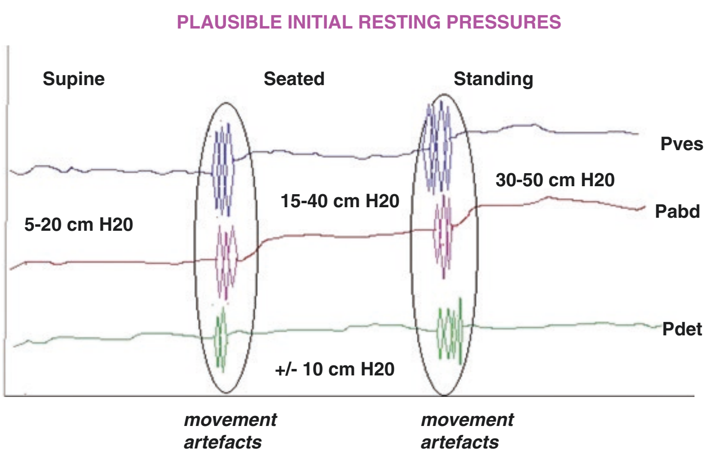


FIGURE 4.13 Plausible ranges for initial values of Pves and Pabd. Resting pressure must be updated when the patient moves from supine to standing position in relation to the position of the pressure transducers

  - If only one pressure is outside the range, the correct zeroing of the relevant transducer should be checked, and then the catheter should be flushed and, in case, repositioned.
- Control of quality of signals
  Before and during the exam, it is necessary to verify that both pressures are registering by asking the patient to cough. Usually, this is done every 50 mL of filling. The amplitude of Pves and Pabd should be similar. If the height of one cough peak is less than 70% of the other, the line with the lower value should be flushed with water and the cough test repeated. Vital signs such as respiration, talking of the patient, and movement should be visible in Pves and Pabd throughout the entire examen as a sign of good quality of pressure signals (Fig. 4.14) (Box 4.1).

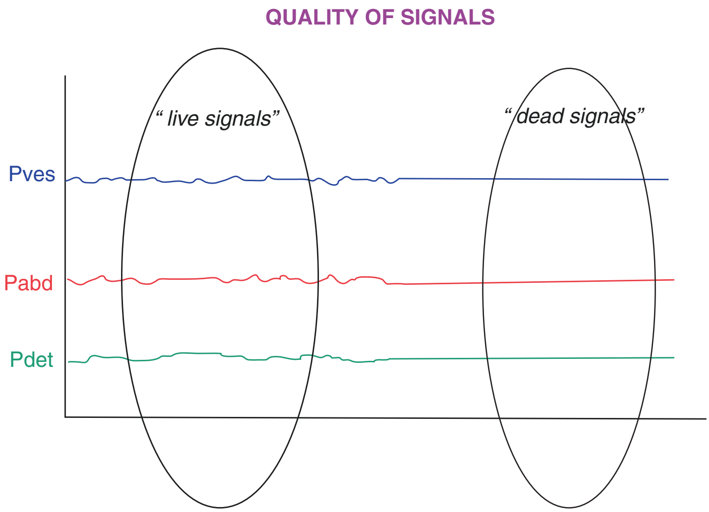


FIGURE 4.14 Pressure fluctuations within 10 cmH_2O of initial resting pressure are considered normal "live signals." The absence of the fine structure of a pressure trace can indicate an error in pressure transmission

Box 4.1 Urodynamic Testings: Key Points for Clinical Practice

1. Detailed clinical evaluation.
2. Informed consent.
3. Room intended for quiet and protected from unnecessary interruptions.

Noninvasive

4. Free uroflowmetry when the patient feels a normal desire to void.
5. Assessment of PVR through ultrasounds or catheter.

Conventional

6. Catheters positioned in *supine* position:
   - 5 Fr rectal balloon catheter inserted at the anus
   - 7 Fr double-lumen transurethral catheter inserted into the urethra
   - EMG surface electrodes attached at both sides of the anus
7. Good urodynamics is carried out by making pressure measurements relative to atmospheric pressure. This is achieved in a water-filled system by placing the pressure transducers at the upper level of the symphysis pubis and by zeroing the equipment with the transducers closed off to the patient and open to the atmosphere.
8. Catheters should be properly connected to the external transducers avoiding any leakage of fluid and flushed by the air.
9. Filling and voiding cystometry are usually conducted with the patient in *sitting* position.
10. Check that resting values for abdominal, intravesical, and detrusor pressures are in a normal range.
11. Prior to bladder filling, signal control quality is done: cough is used to ensure that abdominal and intravesical pressure signals respond equally.

(continued)

Box 4.1 (continued)

12. The bladder is filled at a rate of 50 mL/min.
13. During filling quality control of the signal is checked asking patient to cough. Any artifacts should be immediately corrected.
14. Using annotation marks while running the test is helpful.

4.5 Post-test Patient Care

Most people have urodynamic tests without experiencing any problems. Drinking a large amount of fluid for 24 h is usually enough to avoid mild voiding problems. Anti-inflammatory drugs may be used to resolve burning during micturition. Routine antibiotics are unnecessary and usually not recommended.

Risk factors accounting for a potential increase of UTI following UDS for which a periprocedural antibiotics are advisable include:

- Patients with relevant neurogenic lower urinary tract dysfunction
- Patients with bladder outlet obstruction and/or elevated post-void residual
- Advanced age (older than 70 years)
- Asymptomatic bacteriuria
- Patients with chronic catheters
- Immunosuppression, corticosteroids, and inherent immune deficiency
- Orthopedic implant

Diabetes, menopausal status, history of recurrent UTI, and cardiac vascular disease do NOT require routine antibiotic prophylaxis.

The first choice for prophylaxis is a single oral dose of trimethoprim-sulfamethoxazole before UDS, with alternative antibiotics chosen in case of allergy or intolerance.

Suggested Reading

Overviews

Rosier PFWM, Schaefer W, Lose G, Goldman HB, Guralnick M, Eustice S, et al. International continence society good urodynamic practices and terms 2016 (ICS-GUP2016): urodynamics, uroflowmetry, cystometry and pressure-flow study. Neurourol Urodyn. 2017;36:1243–60.

Leitner L, Walter M, Sammer U, Knüpfer SC, Mehnert U, Kessler TM. Urodynamic investigation: a valid tool to define normal lower urinary tract function? PLoS One. 2016;11:e0163847.

Raz O, Tse V, Chan L. Urodynamic testing: physiological background, setting-up, calibration and artefacts. BJU Int. 2014; 114:22–8.

Gammie A, Clarkson B, Constantinou C, Damaser M, Drinnan M, Geleijnse G, et al. International continence society guidelines on urodynamic equipment performance. Neurourol Urodyn. 2014;33:370–9.

Rosier PF, Giarenis I, Valentini FA, Wein A, Cardozo L. Do patients with symptoms and signs of lower urinary tract dysfunction need a urodynamic diagnosis? ICI-RS 2013. Neurourol Urodyn. 2014;33:581–6.

Winters JC, Dmochowski RR, Goldman HB, Herndon CD, Kobashi KC, Kraus SR, et al. Urodynamic studies in adults: AUA/SUFU guideline. J Urol. 2012;188:2464–72.

Haylen BT, de Ridder D, Freeman RM, Swift SE, Berghmans B, Lee J, et al. An International Urogynecological Association (IUGA)/International Continence Society (ICS) joint report on the terminology for female pelvic floor dysfunction. Neurourol Urodyn. 2010;29:4–20.

Schäfer W, Abrams P, Liao L, Mattiasson A, Pesce F, Spangberg A, et al. Good urodynamic practices: uroflowmetry, filling cystometry, and pressure flow studies. Neurourol Urodyn. 2002;21:261–74.

Abrams P, Cardozo L, Fall M, Griffiths D, Rosier P, Ulmsten U, et al. The standardisation of terminology in lower urinary tract function: report from the standardisation subcommittee of the International Continence Society. Urology. 2003;61:37–49.

Pressure Measurement

Abrams P, Damaser M, Niblett P, Rosier PFWM, Toozs-Hobson P, Hosker G, et al. Air filled, including “air-charged,” catheters in urodynamic studies: does the evidence justify their use? Neurourol Urodyn. 2017;36:1234–42.

Gammie A, Abrams P, Bevan W, Ellis-Jones J, Gray J, Hassine A, et al. Simultaneous in vivo comparison of water-filled and air-filled pressure measurement catheters: Implications for good urodynamic practice. Neurourol Urodyn. 2016;35:926–33.

Digesu GA, Derpapas A, Robshaw P, Vijaya G, Hendricken C, Khullar V. Are the measurements of water-filled and air-charged catheters the same in urodynamics? Int Urogynecol J. 2014;25:123–30.

Quality Control

MacLachlan LS, Rovner ES. Good urodynamic practice: keys to performing a quality UDS study. Urol Clin North Am. 2014;41:363–73.

Hogan S, Jarvis P, Gammie A, Abrams P. Quality control in urodynamics and the role of software support in the QC procedure. Neurourol Urodyn. 2011;30:1557–64.

Gammie A, Drake M, Swithinbank L, Abrams P. Absolute versus relative pressure. Neurourol Urodyn. 2009;28:468.

Patient Care After Tests

Cameron AP, Campeau L, Brucker BM, Clemens JQ, Bales GT, Albo ME, et al. Best practice policy statement on urodynamic antibiotic prophylaxis in the non-index patient. Neurourol Urodyn. 2017;36:915–26.

Okorocha I, Cumming G, Gould I. Female urodynamics and lower urinary tract infection. BJU Int. 2002;89:863–7.

Chapter 5
Interpretation of Tracings and Identification of Artifacts

5.1 Background

Correct interpretation of tracings and recognition of artifacts in the pressure signals are critical for an accurate diagnosis. A number of reports describe the interobserver variation and short-term (i.e., within the session), intermediate-term, and long-term reproducibility of urodynamic investigation. A test–retest variation of 10–15% for various urodynamic parameters has been reported. Basically, this variation can be regarded as the physiological variation of lower urinary tract function, but there is also some evidence that some parameters may be observer-dependent. Therefore, application of standard techniques and continuous quality control is of the greatest significance. While the value of a well-done urodynamics assessment is well established, comparatively few clinicians have received formal training in the area. The familiarity with the most relevant UDS parameters to analyze is of paramount importance to facilitate the correct interpretation of tracings and improve the performance of examinations in daily practice.

G. Vignoli, *Urodynamics for Urogynecologists*,
https://doi.org/10.1007/978-3-319-74005-8_5

5.2 Conventional (Invasive) UDS: Relevant Parameters

5.2.1 Cystometry

The principal aim of cystometry is to reproduce patient storage symptoms (frequency, urgency) and relate them to specific urodynamic events.

Several parameters are recorded during the filling phase including (Fig. 5.1):

- Bladder sensation
- Bladder compliance
- Maximum cystometric capacity
- Detrusor function

5.2.1.1 Bladder Sensation

Bladder sensation during urodynamics is usually recorded by the simple method of asking patients to inform the investigator when they experience different sensations. This is an ICS standardized but subjective measurement with evident limits.

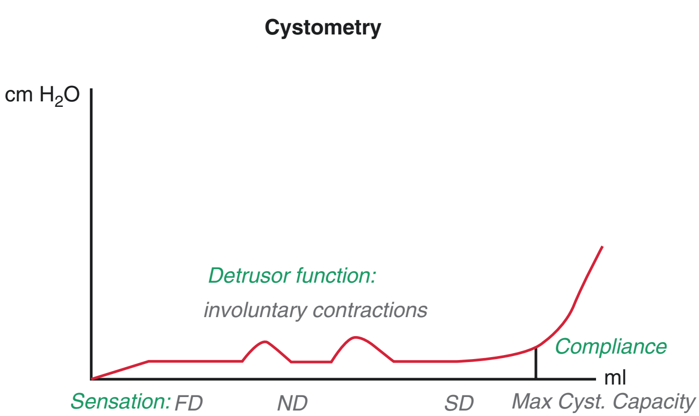


FIGURE 5.1 Parameters monitored during cystometry

Note: The need of strategies to record bladder filling sensations in a more automated and objective way is widely recognized. Toward this end, a keypad "urge score" device was designed to measure sensations during bladder filling.

To date, however, the device has never found a wide use into clinical practice.

In normal subjects, three types of sensations can be observed:

(a) The first sensation of bladder filling (FS), which is felt when the subject becomes aware of bladder filling (it is perceived as a vague sensation in the lower pelvis, which waxes and wanes, and could be easily ignored for few minutes). Usually is felt at 40% of bladder capacity
(b) First desire to void (FD), a constant sensation felt in the lower abdomen that gradually increases with bladder filling and would lead the patient to void in the next convenient moment, but still voiding can be delayed. Usually is felt at 60% of bladder capacity
(c) Strong desire to void (SD), a persistent desire to void felt in the perineum or urethra, without fear of leakage. Usually is felt at 90% of bladder capacity

Note: Volumes of filling sensations and cystometric capacity have to be weighed against the information from the patients' voiding diary, since the laboratory situation may cause a variation in the patient's usual lower urinary tract behavior.

5.2.1.2 Bladder Compliance

Bladder compliance is the relationship between changes in volume and pressure. It is calculated by dividing change in volume by change in Pdet and is expressed in milliliter/centimeter H_2O. Normal bladder compliance values vary between 30 and 100 mL/cmH_2O and are higher in women than in men (usually >40 mL/cmH_2O).

The ICS recommends two standard points for measuring bladder compliance: detrusor pressure at empty bladder and

at maximum bladder capacity or immediately before the start of any detrusor contraction that causes significant leakage.

Bladder compliance in women is considered abnormal if it is below 40 mL/cmH_2O. Values lower that 40 mL/cmH_2O in neurological patients have been associated with a high risk of upper urinary tract complications.

Note: A rule of thumb is that a detrusor pressure of 10 cmH_2O or less at expected bladder capacity for age is acceptable.

5.2.1.3 Maximum Cystometric Capacity

Maximum cystometric capacity is the bladder volume at the end of bladder filling when patients have a strong desire to void and can no longer delay micturition. Normal cystometric capacity varies widely, but is normally between 300 and 500 mL, with lower values in women than in men.

Note: Usually there is a discrepancy in bladder capacity between voiding diary and urodynamics study since in daily life the bladder is usually emptied before a strong or even a "normal" desire to void is felt on cystometry.

5.2.1.4 Detrusor Function

Detrusor function during filling cystometry can be classified in:

(a) *Normal*, which allows filling with little or no change in pressure and no involuntary contractions despite provocation (usually cough)
(b) *Detrusor overactivity (DO)*, which is the urodynamic observation of involuntary detrusor contraction during bladder filling that may be spontaneous or provoked by cough, postural changes, or rapid filling

Typically, there are two patterns of DO (Fig. 5.2):

(a) *Phasic DO*, defined by the presence of fluctuating waveform, which may or may not lead to urinary incontinence

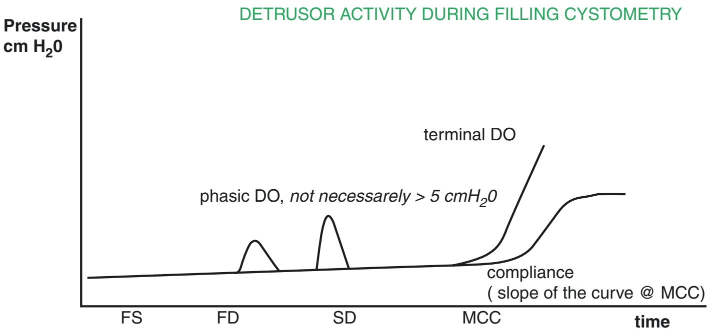


FIGURE 5.2 Patterns of DO

Originally, ICS stated that, in order to diagnose "detrusor overactivity," the contraction should be at least 15 cmH_2O. However, it was subsequently realized that involuntary detrusor contractions of <15 cmH_2O could cause significant symptoms. In practice, there is no standardized minimum value for detrusor contractions though it may be difficult to be certain whether an involuntary contraction has occurred if the phasic wave is <5 cmH_2O.

(b) *Terminal DO*, classified as a single involuntary detrusor contraction occurring at cystometric capacity, which cannot be suppressed and often results in complete bladder emptying

Note: The incidence of detrusor overactivity is higher when the patient is in the sitting position during cystometry, rather than the supine position.

In summary, during filling phase the bladder should have constantly low pressure that usually does not reach more than 6–10 cmH_2O above baseline at the end of filling (end-filling pressure), and there should be no involuntary contractions, with normal first sensation ranging between 100 and 250 mL, bladder compliance between 30 and 100 mL/cmH_2O, and maximum bladder capacity between 450 and 550 mL (see Box 5.1).

Box 5.1: Urodynamic Parameters During Filling Phase in Women

Pdet	6–10 cmH_2O
FS	100–250 mL
MCP	450–550 mL
Compliance	>40 mL/cmH_2O

No involuntary contractions

5.2.2 *Leak Point Pressures*

During the filling phase are also evaluated the leak point pressures.

There are currently two types of leak point pressures that measure different functional areas of the lower urinary tract:

- Detrusor leak point pressure—bladder
- Abdominal leak point pressure—urethra

Detrusor leak point pressure (DLPP) is defined by the ICS as the lowest Pdet at which urine leakage occurs in the absence of either detrusor contraction or increased abdominal pressure. The rise in bladder pressure is secondary to low bladder compliance. This value reflects resistance that the urethra offers to the bladder, mainly by the action of the external sphincter (Fig. 5.3).

In patients with neurogenic bladder, a high DLPP can threaten upper urinary tract function: a DLPP ≥40 cmH_2O is usually associated with upper tract damage if not treated. The bladder volume at which detrusor pressure equals 40 cmH_2O is considered the patient's "safe" bladder capacity.

Abdominal leak point pressure (ALPP) or Valsalva leak point pressure (VLPP) is intravesical pressure at which urine leakage occurs because of increased abdominal pressure in the absence of detrusor contraction. It measures the ability of the urethra to resist an increase in abdominal pressure. ALPP is usually tested during cystometry after the bladder has been

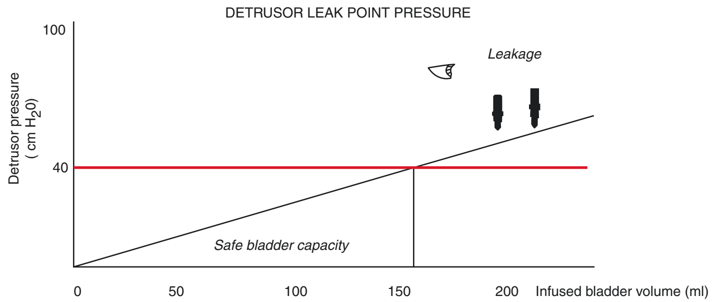


FIGURE 5.3 Detrusor leak point pressure: the lowest intravesical pressure at which leakage is noted around the catheter. The bladder volume at which detrusor pressure equals 40 cmH_2O is considered the maximal "safe" bladder capacity

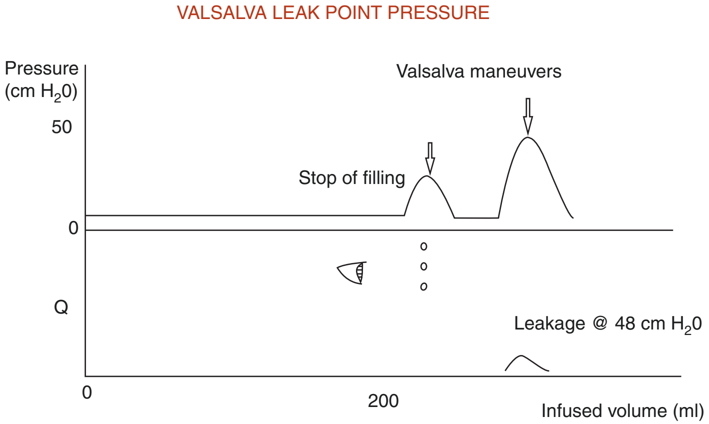


FIGURE 5.4 Leakage occurring during Valsalva maneuver at 200 mL of filling

filled to at least 150–200 mL. The patient is then asked to do cough or a Valsalva maneuver until she leaks. VLPP is the lowest pressure at which incontinence occurs. The procedure is facilitated by having one person observing for leakage ad one person observe for pressure since only consistent leakages can be recorded by the flowmeter (Fig. 5.4).

Box 5.2: Leak Point Pressures

- DLPP not suitable in normal females.
- With reduced bladder compliance (lower than 40 mL/cmH_2O), a DLPP>40 cmH_2O is considered dangerous for upper urinary tract. Bladder volume with pressure below 40 cmH_2O is considered as "safe bladder capacity."
- ALPP not suitable in normal females.
- *In incontinent women*:
  - ALPP lower than 60 cmH_2O indicates an ISD.
  - ALPP greater than 100 cmH_2O indicates urethral hypermobility.
  - Values between 60 and 100 cmH_2O are suggestive of both ISD and hypermobility.

In normal women, no incontinence should be recorded, whatever the increase in abdominal pressure.

In incontinent women the test correlates with severity of incontinence and may indicate the presence an intrinsic sphincteric deficiency (ISD). A value of ALPP greater than 100 cmH_2O is usually associated with urethral hypermobility. A value lower than 60 cmH_2O is usually associated with ISD. Values between 60 and 100 cmH_2O are suggestive of both ISD and hypermobility (see Box 5.2).

5.2.3 Pressure/Flow Study

Voiding phase starts when "permission to void "is given or when uncontrollable voiding begins and ends when the patient considers voiding has finished. Voiding is usually assessed by measuring urine flow rate and voiding pressures. Pressure/flow study or voiding cystometry is the method by which the relationship between pressure in the bladder and urine flow rate is measured during bladder

emptying. Pressure/flow study is considered to be the gold standard for quantifying and grading bladder outlet obstruction (BOO), and differentiating between BOO and detrusor underactivity (DU) Pdet at maximum flow and maximum flow (Qmax) are the most significant parameters. Their values can be plotted on pressure flow nomograms to classify patients as being either obstructed or not obstructed and, at the same time, grade the severity of obstruction.

Different types of nomograms have been developed for male, including ICS nomogram, Abrams–Griffiths nomogram, and Schafer nomogram.

However, since male and female have unique voiding characteristics, these nomograms do not apply to female.

The only nomogram currently used for the analysis of P/Q in women is that described by Groutz and Blaivas (Fig. 5.5). The nomogram consists of four zones, which classify patients into four categories: zone 0 (normal or no obstruction), zone 1 (mild obstruction), zone 2 (moderate obstruction), and zone 3 (severe obstruction). Two parameters are needed to construct this nomogram: Free Qmax and Pdet.max. Free Qmax is preferred to Qmax during P/Q because Pdet at Qmax and Qmax cannot be evaluated if the patient does not void during the test. The nomogram does not include the assessment of bladder contractility.

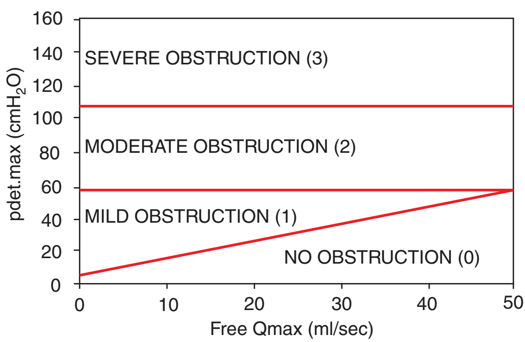


FIGURE 5.5 The Groutz and Blaivas nomogram

Box 5.3: Urodynamic Parameters During Voiding Phase in Women

Voiding by urethral relaxation	60%
Voiding by abdominal straining	20%
Voiding by detrusor contraction	20%
Qmax	13–25 mL/s
Pdet at Qmax	18–30 cmH_2O
Pdet max	22–46 cmH_2O
VV	250–650 mL
Mild obstruction	Pdet max: 10–60 cmH_2O/ Qmax<12 mL/s
Moderate obstruction	Pdet max: 60–110 cmH_2O/ Qmax<12 mL/s
Severe obstruction	Pdet max: 110–160 cmH_2O/ Qmax<12 mL/s

As mentioned before (Chap. 1), 60% of females void by pelvic floor relaxation only without any increase in detrusor pressure, 20% utilizes abdominal straining, and the other 20% void by detrusor contraction like male.

Qmax ranges from 13 to 25 mL/s, Pdet at Qmax ranges from 18 to 30 cmH_2O, Pdet.max ranges from 22 to 46 cmH_2O, and voided volume (VV) ranges between 250 and 650 mL (see Box 5.3).

Note: The majority of female patients are able to void relatively well provided the catheters are properly fixed. Nevertheless, investigators should interpret pressure–flow voiding parameters and the subsequent post-void residual urine together with the catheter-free voiding parameters,

preferably of multiple flows to avoid misinterpretations due to the obstructive effects of catheter.

5.3 Artifacts Identifications and Remedial Actions

Minimizing all equipment artifacts and ensuring the quality of pressure recording makes the trace easier to interpret and enables clear identification of pathophysiological features.

5.3.1 Uroflowmetry: Knock to the Equipment (Fig. 5.6)

The knock to the equipment is recognized by a spike of less than 2 s over the flow curve.

The artifact is less common in female than in male since female void seated in the commode except in cases of voiding dysfunction when she may decide to void in semi-seated position.

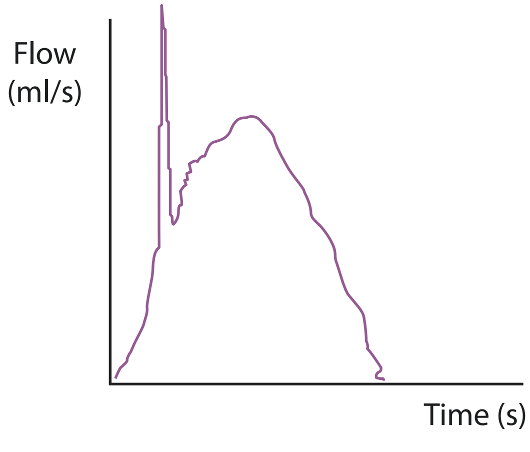


FIGURE 5.6 Spike of less than 2 ms over the flow curve due to an accidental knock to the equipment

5.3.2 *Pressure Traces*

The most common pressure trace artifacts include:

(a) Poor pressure transmission—(damping of the signal due to air bubbles in the system) (Fig. 5.7)

Accurate and high-quality pressure transmission is essential during urodynamic examination. The definition of good pressure transmission is that changes in intraabdominal pressure should be recorded equally on both Pves and Pabd, with the result that Pdet is unchanged. In most of the cases, poor pressure transmission is a consequence of air bubbles in the system with a damping of the signal.

If pressure spikes are of unequal height, the catheter with the smaller spike is to be flushed, and the patient is asked to provide a cough signal again. If the problem persists, the affected catheter should be checked for problems.

Note: Once the test is underway, cough signal spikes are used every minute to assess the quality of pressure recording and to take any necessary corrective steps as problems occur.

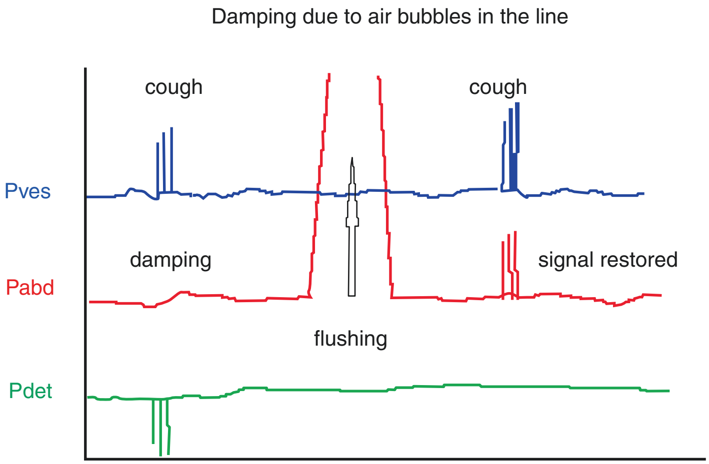


FIGURE 5.7 Poor quality signal in Pabd with Pdet negative. Following flushing of the line, pressure transmission is restored

A lack of regular cough signals and failure to correct a problem once it has occurred can lead to traces being rejected on quality base.

(b) Pressure line descent—leakage (Fig. 5.8)

A pressure line descent is usually caused by a leak somewhere between the pressure transducer and patient. It may occur also when the taps are left open to both the syringe and the patient. Initially a leak will affect pressure transmission and may be mistaken for a displaced catheter or air bubble due to the lack of agreement in the height of cough signal peaks between Pves and Pabd. The affected tube should be checked for taps position and leakage, and then it should be flushed to remove any trapped air and restore transmission of pressure.

(c) Negative fluctuations of Pdet—rectal contractions (Fig. 5.9)

Rectal contractions may cause temporary increases in the Pabd reflecting negatively in Pdet, without any change in

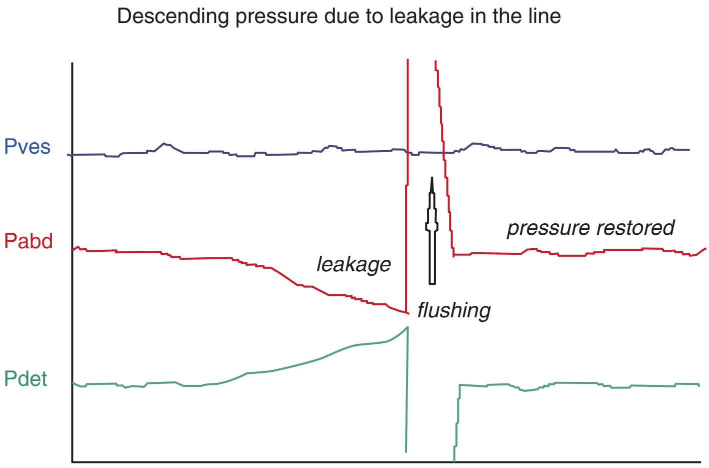


FIGURE 5.8 Leakage in Pabd line. Remedial action consists in checking the site of leakage. Then the line should be flushed to remove air bubbles and restore good quality transmission of pressure

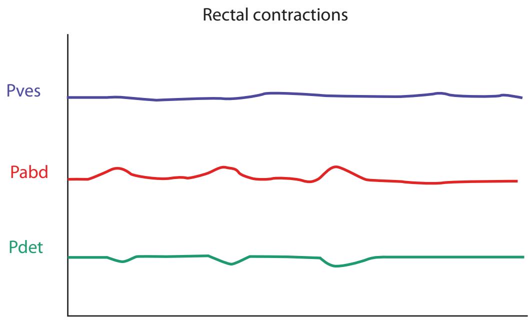


FIGURE 5.9 The peaks in Pabd due to rectal contractions cause negative waves in Pdet

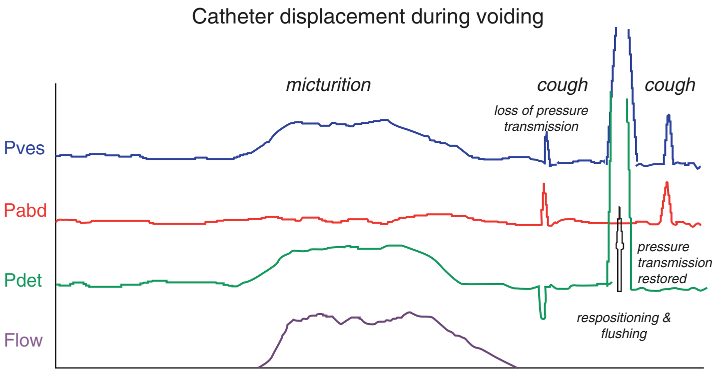


FIGURE 5.10 Displaced catheter result in poor quality cough signal in Pves after voiding. After repositioning of the catheter and flushing, good quality pressure transmission is restored

Pves. Sometimes rectal contractions can be mistaken for DO, as wavelike pressure changes will occur on the Pdet line, although there is no change in Pves which would occur in DO.

There are no remedial actions, but care should be taken not to identify such Pdet decreases as DO.

(d) Displaced catheter during voiding (Fig. 5.10)

If a catheter moves down into either the urethral or anal sphincter region, the resting pressure may increase, usually

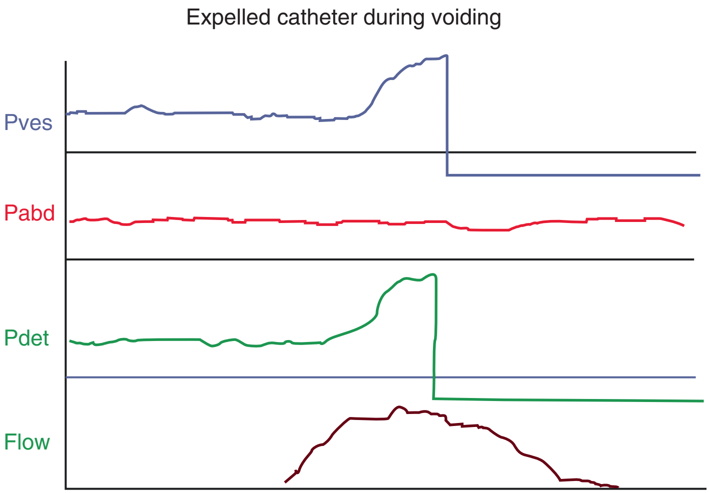


FIGURE 5.11 Sharp drop in Pves and Pdet pressures indicate that vesical catheter has been expelled during micturition

with reduction in pressure transmission. Catheter movement is recognized by assessing pressure transmission after voiding. If there is poor pressure transmission post void, care must be taken when using the pressure readings recorded during the void, as their validity may be unreliable.

(e) Expelled catheter (vesical or rectal/vaginal) at void (Fig. 5.11)

When a catheter is expelled, live signal is lost, and pressure drops abruptly, usually to an implausible pressure value. The significance of an expelled catheter depends on whether the urodynamic question has been answered before the catheter is expelled.

If the urodynamic question cannot be answered because of the expelled catheter, a new one should be inserted, the patient refilled, and the voiding phase repeated.

Note: The reported descriptions are all with reference to water-filled pressure measurement systems, although some of these artifacts may occur in other types of recording system as well.

FIGURE 5.12 Alternating current (60 Hz) artifact. The noise presents at exact frequency of 60 Hz, as its name indicates

5.3.3 EMG: 60 Hz artifact (Fig. 5.12)

A 60 Hz or alternating current artifact is the most common ambient noise during EMG recording. The problem arises when the impedance of the active electrode becomes inadequate. In this situation, the ground becomes an active electrode that, depending on its location, produces the 60 Hz artifact. Essentially any device (computer, radio, lamp, etc.) that is plugged into the wall AC (alternating current) outlet may emit ambient noise.

A 60 Hz frequency from power line may overlay or cancel the signal being recorded from the muscle.

Remedial action include switching-off any possible source of interference (power line, phone line, radio waves) and checking the electrode-to-skin contact including the ground (poor skin prep, too little conducting gel).

Suggested Readings

Interpretation of Tracings

D'Ancona CA, Gomes MJ, Rosier PF. ICS teaching module: cystometry (basic module). Neurourol Urodyn. 2017;36:1673–6.

Tarcan T, Demirkesen O, Plata M, Castro-Diaz D. ICS teaching module: detrusor leak point pressures in patients with relevant neurological abnormalities. Neurourol Urodyn. 2017;36:259–62.

Rosier PF, Kirschner-Hermanns R, Svihra J, Homma Y, Wein AJ. ICS teaching module: analysis of voiding, pressure flow analysis (basic module). Neurourol Urodyn. 2016;35:36–8.

Mahfouz W, Al Afraa T, Campeau L, Corcos J. Normal urodynamic parameters in women: part II—invasive urodynamics. Int Urogynecol J. 2012;23:269–77.
Nager CW, Albo ME, Fitzgerald MP, McDermott S, Wruck L, Kraus S, et al. Reference urodynamic values for stress incontinent women. Neurourol Urodyn. 2007;26:333–40.
Craggs MD. Objective measurement of bladder sensation: use of a new patient-activated device and response to neuromodulation. BJU Int. 2005;96(Suppl 1):29–36.

Artifacts Recognition

Gammie A, D'Ancona C, Kuo H-C, Rosier P. ICS teaching module: Artefacts in urodynamic pressure traces (basic module). Neurourol Urodyn. 2017;36:35–6.
Hogan S, Gammie A, Abrams P. Urodynamic features and artefacts. Neurourol Urodyn. 2012;31:1104–17.

Chapter 6
Supplementary Urodynamic Tests

6.1 Background

In 2016 the ICS Standardisation Steering Committee has published a report to update the International Continence Society's Good Urodynamic Practice 2002 (GUP2002) with the aim of including new evidence and information on urodynamic practice and urodynamic quality control and the revised ICS standard on urodynamic equipment. The ICS-GUP2016 makes a distinction between the ICS standard urodynamic test including uroflowmetry and PVR plus transurethral cystometry and pressure-flow study and ICS supplementary urodynamic test including urethral profilometry, EMG and video. Ambulatory urodynamics monitoring has not been further discussed in the new report and still refers to ICS 2000 standards.

6.2 Urethral Profilometry

The urethral pressure profilometry (UPP) is a graph indicating changes in intraluminal pressure along the length of the urethra.

UPP quantifies the occlusive pressure generated by active (smooth and striated muscles) and passive structures (spongy mucosal layer and fibroelastic connective tissue) and of the

G. Vignoli, *Urodynamics for Urogynecologists*,
https://doi.org/10.1007/978-3-319-74005-8_6

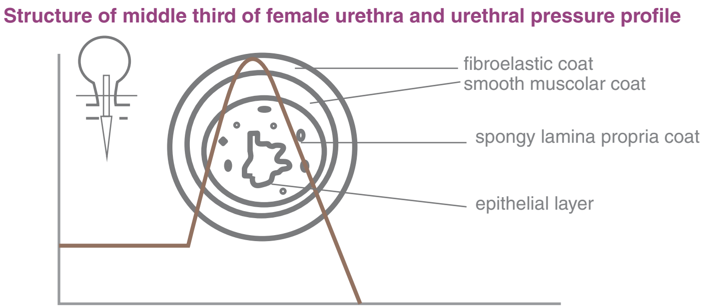


FIGURE 6.1 Structure of female urethra with superimposed urethral pressure profile

urethra allowing the evaluation of urethral competence (Fig. 6.1). Two variations of this measurement are commonly reported:

- UPP at rest
- UPP under cough with the assessment of pressure transmission ratio

6.2.1 Technical Notes

The three main methods for UPP measurement are:
- Perfused catheters
- Microtip catheters
- Air-filled balloon catheters

The technique of perfused catheters is the most popular and is mainly based on the description by Brown and Wickham. The perfusion rate should be 2 mL/min and withdrawal rate of 1 mm/s for an accurate measurement of closure pressure. Perfusion rates lower than 2 mL/min usually fail to record the true urethral pressure unless the withdrawal rate is extremely slow. Withdrawal rate is irrelevant with the stress profile since the % PTR is assessed in specific points (usually

each half centimetre) from the bladder neck to the external meatus.

Because of the rapid frequency response, microtip catheters are particularly suitable for recording very rapid changes in intra-urethral pressure, such as those occurring during cough.

However, their stiffness may lead to an interaction between the transducer and the urethral wall resulting in a directional artefact. The results obtained with the transducer in the lateral orientation (3 or 9 o'clock) compared with the anterior position seems to be more reliable.

Air-filled catheters (AFCs) have been actively marketed for the past few years since they seem *faster and simpler to set up than any other technology.*

Studies comparing measurement of urethral pressure by air-filled catheters with measurement by microtip catheters have indicated that the values obtained are statistically significantly different but are more reproducible with the air-filled systems.

Compared with water-filled catheters, air-filled catheters act as an overdamped system less sensitive to artefacts but lower in response. *In addition*, the air-filled catheters consistently produce higher mean pressure values than the water-filled catheters. Therefore, the techniques are not interchangeable, and caution must be used when comparing urodynamic parameters using air-charged and water-filled catheters.

6.2.2 UPP at Rest

Parameters recorded in the resting UPP include (Fig. 6.2):

- Maximum urethral pressure
- Maximum closure pressure
- Functional length

Reference values are reported in the Box 6.1.

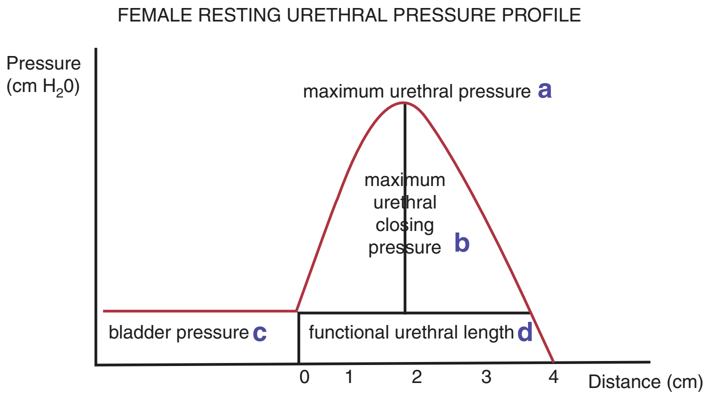


FIGURE 6.2 Diagram of female resting pressure profile indicating (**a**) maximum urethral pressure, (**b**) maximum urethral closure pressure, (**c**) intravesical pressure, (**d**) functional profile length

Box 6.1: Magnitude of Reference Values of Static UPP

Parameter	*Value*
Maximum urethral pressure (MUP)	50–80 cm H_2O
Maximum urethral closing pressure (MUCP)	40–70 cm H_2O
Functional length	3–4 cm

It is well established that in women the UPP changes with age. MUP, MUCP and the urethral length increase from infancy to the age of 20–25 years. Thereafter, a gradual decline in the MUCP of approximately 15% per decade can be observed (Fig. 6.3).

Both MUP and MUCP are reduced in stress-incontinent women. However, since the overlap between the values from continent and incontinent women is substantial, it is impossible to define a cut-off level that allows differentiation between women with and without stress incontinence.

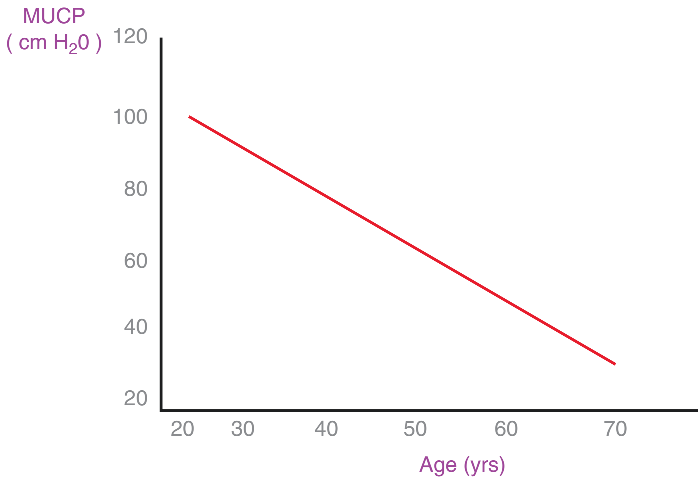


FIGURE 6.3 MUCP against age group

An MUCP value less than 20 cm H_2O is the most popular single predictor of ISD and has been considered suggestive of poor outcome of conventional antiincontinence surgery.

An MUCP value greater than 75 cm H_2O may be indicative of sphincter overactivity and in presence of an interrupted flow may be suggestive of a dysfunctional voiding.

Several studies have shown considerable test-retest variation of all urethral pressure. Measurements or parameters and those normal and pathological values of urethral pressure parameters are largely overlapping.

Due to poor sensitivity and specificity and significant test-retest variation, urethral profilometry is not recommended as the only measurement in females with urinary incontinence, but it should be judged in relation to other urodynamic tests (i.e. urodynamic stress incontinence, Valsalva leak point pressure) and physical examination.

6.2.3 Stress UPP

Stress UPP measures the rise in intra-abdominal pressure transmitted to the proximal urethra. In normal women without urethral hypermobility, increases in intravesical pressure and proximal urethral pressure should be similar (Fig. 6.4).

The pressure transmission ratio is a different parameter, recording the increment of urethral pressure under cough as a percentage of intravesical pressure elevation. Pressure transmission ratio (% PTR) decreases progressively from the bladder neck to the external meatus. In normal women, its value should exceed 90% in the proximal urethra.

Values lower than 90% indicates urethral hypermobility, but do not necessarily indicate incontinence (Box 6.2).

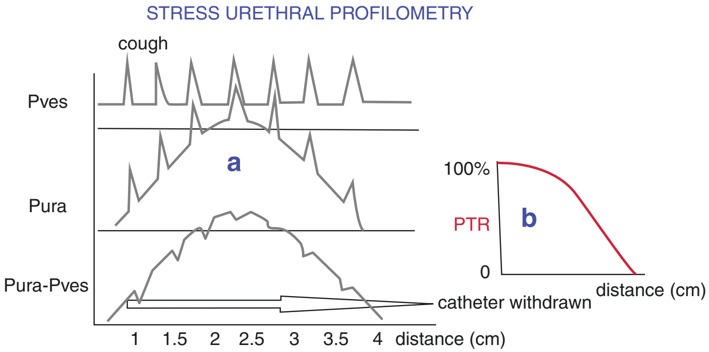


FIGURE 6.4 Diagram of female stress urethral pressure profile indicating Pves, Pura and Pura minus Pves under cough (**a**). Pressure transmission ratio is calculated as Pura/Pves × 100 along the functional urethral length divided in quartiles and decreases from bladder neck to the external meatus (**b**)

Box 6.2: Urethral Profilometry

- MUCP unsuitable for diagnosis of SUI
- MUCP below 20 cm H_2O raises the possibility of ISD
- MUCP values above 75 cm H_2O for women are considered hypertonic and suggestive of dysfunctional voiding
- %PTR <90% indicates hypermobility, but not incontinence

Note: Current urethral function tests are only very modestly suited to further "subcategorize" patients with stress predominant urinary incontinence for treatment or to judge the severity of incontinence. There is conflicting evidence that low urethral closure pressures are associated with poorer success rates of retropubic and transobturator midurethral slings. On the other hand, the surgical procedures that are more "obstructive" (retropubic vs. transobturator slings) increase the risk of de novo overactive bladder syndrome.

6.3 Electromyography

Sphincter EMG records bioelectric potentials generated during muscle depolarization allowing clinicians to completely evaluate the striated sphincter complex and pelvic floor activity during bladder filling and voiding.

Clinically, the most important information obtained from sphincter EMG is coordination or discoordination between the external urethral sphincter (EUS) and the bladder. Investigation may be performed with a variety of electrodes including surfaces, needle and wire electrodes. Surface electrodes (the most

commonly employed) are placed in the buttock close to the anus. Needle and wire electrodes are placed lateral to the urethral meatus and advanced parallel to the urethra to a distance of about 1–2 cm. Unlike surface electrodes, they are positioned in the muscle of interest, allowing the detection of activity in individual motor units. In addition, since wire electrodes are self-retaining, they are preferable when one expect changes in patient position during the examination.

Normally, EMG activity from the EUS is low at rest. It intensifies as fluid volume in the bladder grows, during bladder filling, due to EUS contraction. It is known as the "guarding reflex". During voiding, EMG activity disappears completely for a few seconds before detrusor contraction starts. Once the bladder is empty, EMG activity resumes. With any increase of abdominal pressure (cough, straining), there is an increase of EMG activity proportional to the level of stress (Fig. 6.5).

Clinically, the most important information obtained from sphincter EMG is coordination or discoordination between

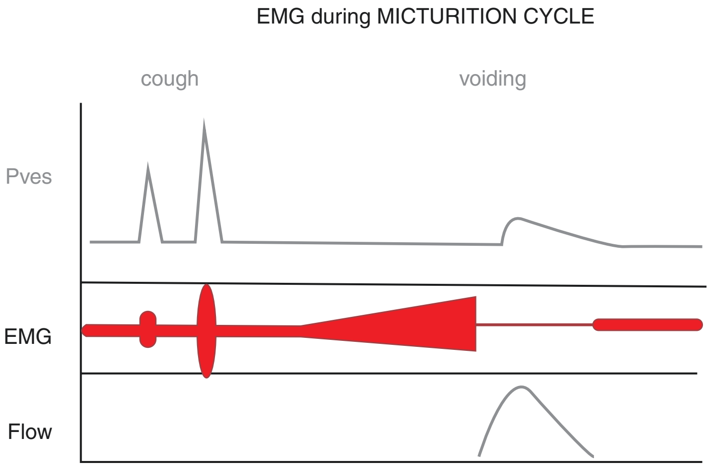


FIGURE 6.5 EMG activity during micturition cycle. During bladder filling, there is a gradual increase in sphincter EMG activity. During cough there is a reflex contraction of the sphincter to prevent incontinence. This process is organized by urethral reflexes known collectively as the "guarding reflex". During voiding the sphincter EMG tracing becomes silent

Box 6.3: Electromyography

- EMG activity increases progressively during bladder filling.
- EMG activity increases with any increase of abdominal pressure (cough, straining).
- EMG activity is silent during voiding.
- Persistent EMG activity during voiding is termed detrusor sphincter dyssynergia in neurologic patient.
- Persistent EMG activity during voiding in neurologically normal subjects is termed dysfunctional voiding.

the external urethral sphincter (EUS) and the bladder. Failure of the sphincter to relax or stay completely relaxed during micturition is abnormal. When it occurs in patients with neurologic disease, it is termed detrusor sphincter dyssynergia. The term detrusor sphincter dyssynergia cannot be used in the absence of neurologic disease. In these conditions, the preferred term is dysfunctional voiding (Box 6.3).

6.4 Videourodynamic

Videourodynamic combines simultaneously anatomical and functional information (Fig. 6.6). For many years, videourodynamic (VUD) has been considered the gold standard for the functional evaluation of lower urinary tract.

The importance of VUD is based upon the concept that synchronous visualization of anatomy of lower urinary tract and assessment of function may give more accurate diagnoses. Anatomical observations, during filling phase, include bladder neck closure at rest and during stress, its position in relation to the pubis symphysis, bladder wall morphology and any diverticula and vesico-ureteral reflux (VUR). During voiding phase, VUD allows the distinction of bladder neck versus external sphincter dyssynergia in neurologic patients and accurate localization of obstruction after mid-urethral sling

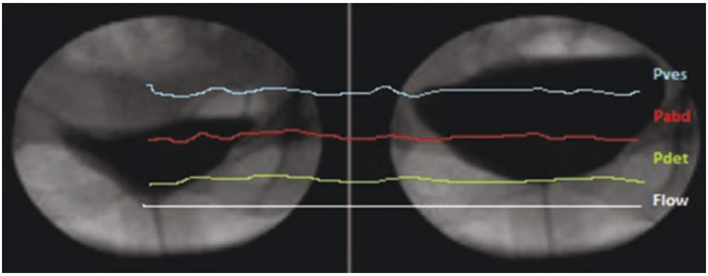


FIGURE 6.6 Videourodynamics. The bladder is filled with a radio-opaque contrast medium, with the simultaneous measurement of intravesical pressure and intra-abdominal pressure. The subtracted detrusor pressure is calculated automatically and flow is recorded with the flowmeter. Pressure measurements and accompanying radiographic pictures are mixed together allowing subsequent review and analysis

insertion. In a broader context, VUD has been advocated in situations in which UD alone fails to provide sufficient diagnostic information to guide therapy, especially in patients with relevant neurological diseases.

In the recent years, it has become controversial whether VUD is always necessary for every patient. Most of critical questions can often be answered by conventional urodynamics sometime supplemented by a separate voiding cystourethrography (VCUG). The necessity of special equipment and radiation exposure mean that the benefits should be weighed against the potential disadvantages. The basic question is under what circumstances does the anatomic information obtained by video provide either useful or critical information.

Low-grade evidence supports the use of VUD in the follow-up of the patient with NLUTD and potential hazard to the upper urinary tract.

Note: "Unsafe bladder "is defined by a filling pressure greater than 40 cm H_2O, compliance lower than 10 mL/cm H_2O and detrusor leak point pressure greater than 40 cm H_2O.

According to EAU guidelines, in high-risk patients, the upper urinary tract should be checked by creatinine and renal

ultrasound once every 6 months, and VUD should be recommended if either is abnormal.

There is only low-grade evidence for the added value of VUD in non-neurogenic LUTS/LUTD.

In women, the use of VUD has been advocated to distinguish bladder neck dyssynergia (a quite rare condition) from dysfunctional voiding since conventional UD with EMG do not correlate well with visual assessment of urethral opening characteristics.

In the past VUD was recommended for the assessment of female SUI (Blaivas SUI classification), but recent data indicate that it has no impact on surgery outcome.

Recurrent SUI in female is probably the only real indication for VUD.

In recurrent SUI, the assessment of bladder neck and urethral hypermobility is crucial, since secondary procedures have been associated with reduced cure rates in the presence of limited urethral mobility and intrinsic sphincter deficiency. VUD may improve the evaluation of bladder neck and urethral hypermobility facilitating optimal preoperative counselling.

Finally, radiation exposure during VUD (currently 488 ($\pm$336) cGy $\times$ cm^2 with an exposure time of 97.5 ($\pm$34) s) should further be evaluated in order to keep radiation doses as low as reasonably achievable (ALARA). This might be achieved by using the newest techniques such as pulsed fluoroscopy and image area reduction.

6.5 Ambulatory Urodynamics Monitoring (AUM)

Urodynamic investigations may be considered as nonphysiological tests because of several artificial factors, such as privacy issues and patient embarrassment, catheterization, supraphysiological filling rates and patients' immobile position during evaluation.

Ambulatory urodynamics monitoring (AUM) is performed in a similar way to conventional urodynamics but differs in some specific elements: It uses natural filling of the bladder (the patients are usually asked to drink extra), and testing lasts

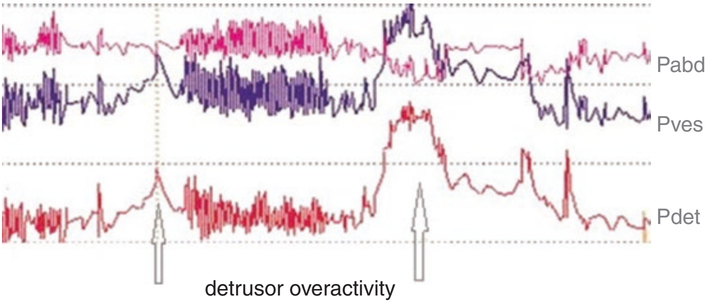


Figure 6.7 Downloaded tracing of AUM indicating the presence of detrusor overactivity

for approximately 2–4 h. Patients are fully dressed after the initiation of the test and are able to leave the urodynamic room, which may reduce anxiety and embarrassment.

The investigation is considered as a supplementary urodynamic test and may be a useful tool to investigate lower urinary tract dysfunction in patients with inconclusive results on laboratory urodynamic testing. The specific technical demands and technical reliability of the investigation are well known, but its clinical sensitivity and specificity are not well established.

In particular, AUM has been showed to be more sensitive than laboratory urodynamics in diagnosing detrusor overactivity (Fig. 6.7), but the level of evidence for this measurement is low. Furthermore, the fact that AUM shows abnormalities of bladder function, especially detrusor overactivity, in healthy volunteers may also be considered a sign of lesser specificity.

Suggested Readings

Urethral Profilometry

Gajewsy J, Rosier P, Rahnama'i S, Abrams P. Do we assess urethral function adequately in LUTD and NLUTD? ICI-RS 2015. Neurourol Urodyn. 2017;36:935–42.

Moe K, Schiøtz HA, Kulseng-Hanssen S. Outcome of TVT operations in women with low maximum urethral closure pressure. Neurourol Urodyn. 2017;36:1320–4.

Klünder M, Sawodny O, Amend B, Ederer M, Kelp A, Sievert KD, et al. Signal processing in urodynamics: towards high definition urethral pressure profilometry. Biomed Eng Online. 2016a;15:31.

Klünder M, Amend B, Vaegler M, Kelp A, Feuer R, Sievert KD. High definition urethral pressure profilometry: evaluating a novel microtip catheter. Neurourol Urodyn. 2016b;35:888–94.

Burden H, Warren K, Abrams P. Leak point pressures: how useful are they? Curr Opin Urol. 2015;25:317–22.

Kapoor D, Housami F, White P, Swithinbank L, Drake M. Maximum urethral closure pressure in women: normative data and evaluation as a diagnostic test. Int Urogynecol J. 2012;23:1613–8.

Mikhail MS, Rosa H, Palan P, Anderson P. Comparison of preoperative and postoperative pressure transmission ratio and urethral pressure profilometry in patients with successful outcome following the vaginal wall patch sling technique. Neurourol Urodyn. 2005;24:31–4.

Trowbridge ER, Wei JT, Fenner DE, Ashton-Miller JA, Delancey JO. Effects of aging on lower urinary tract and pelvic floor function in nulliparous women. Obstet Gynecol. 2007;109:715–20.

Lemack GE. Urodynamic assessment of patients with stress incontinence: how effective are urethral pressure profilometry and abdominal leak point pressures at case selection and predicting outcome? Curr Opin Urol. 2004;14:307–11.

Khullar V, Cardozo L. The urethra (UPP, MUPP, instability, LPP). Eur Urol. 1998;34(Suppl 1):20–2.

Electromyography

Krhut J, Zachoval R, Rosier P, Shelly B, Zvara P. ICS Educational Module Electromyography in the assessment and therapy of lower urinary tract dysfunction in adults. Neurourol Urodyn. 2017;37(1):27–32.

Kirby AC, Nager CW, Litman HJ, Fitzgerald MP, Kraus S, Norton P, Sirls L, Rickey L, Wilson T, Dandreo KJ, Shepherd J, Zimmern P; Urinary Incontinence Treatment Network. Perineal surface electromyography does not typically demonstrate expected relaxation during normal voiding. Neurol Urodyn. 2011;30:1591–6.

Mahajan ST, Fitzgerald MP, Kenton K, Shott S, Brubaker L. Concentric needle electrodes are superior to perineal

surface-patch electrodes for electromyographic documentation of urethral sphincter relaxation during voiding. BJU Int. 2006;97:117–20.

Mayo ME. The value of sphincter electromyography in urodynamics. J Urol. 1979;122:357–60.

Vereecken RL, Derluyn J, Verduyn H. Electromyography of the perineal striated muscles during cystometry. Urol Int. 1975;30:92–8.

Videourodynamic

Peng CH, Chen SF, Kuo HC. Videourodynamic analysis of the urethral sphincter overactivity and the poor relaxing pelvic floor muscles in women with voiding dysfunction. Neurourol Urodyn. 2017;36(8):2169–75.

Anding R, Rosier P, Smith P, Gammie A, Giarenis I, Rantell A, et al. When should video be added to conventional urodynamics in adults and is it justified by the evidence? ICI-RS 2014. Neurourol Urodyn. 2016;35:324–9.

Giarenis I, Phillips J, Mastoroudes H, Srikrishna S, Robinson D, Lewis C, Cardozo L. Radiation exposure during videourodynamics in women. Int Urogynecol J. 2013;24:1547–51.

Nitti VW, Tu LM, Gitlin J. Diagnosing bladder outlet obstruction in women. J Urol. 1999;161:1535–40.

AUM

Digesù A, Gargasole C, Hendricken C, Gore M, Kociancic E, Khulla V, Rosier PF. ICS teaching module: ambulatory urodynamic monitoring. Neurourol Urodyn. 2017;36:364–7.

Salvatore S, Khullar V, Cardozo L, Anders K, Zocchi G, Soligo M. Evaluating ambulatory urodynamics: a prospective study in asymptomatic women. BJOG. 2001;108:107–11.

Radley SC, Rosario DJ, Chapple CR, Farkas AG. Conventional and ambulatory urodynamic findings in women with symptoms suggestive of bladder overactivity. J Urol. 2001;166:2253–8.

van Waalwijk van Doorn E, Anders K, Khullar V, Kulseng-Hanssen S, Pesce F, Robertson A, Rosario D, Schafer W. Standardisation of ambulatory urodynamic monitoring: report of the standardisation sub-committee of the international continence society for ambulatory urodynamic studies. Neurourol Urodyn. 2000;19:113–25.

Chapter 7
UDS in Stress Urinary Incontinence Syndrome (SUI-S)

7.1 Background

There remains no clear consensus as to whether urodynamic testing enhances surgical outcome of stress urinary incontinence treatments by improving case selection or altering the surgical approach based on study findings. As treatment strategies for stress urinary incontinence have developed over the last several years to a more uniform approach, it is less clear that urodynamic testing will influence the choice of surgical technique.

7.2 Types of Urinary Incontinence in Female

The main types of urinary incontinence (UI) in female are stress (SUI), urgency (UUI), and mixed urinary incontinence (MUI). The overall prevalence of UI in female is 19–26%, increasing with age; this is most apparent for urgency urinary incontinence (UUI) and mixed urinary incontinence (MUI), since, interestingly, the prevalence of stress urinary incontinence (SUI) do not seem to increase with age. UI is associated with a negative impact on a woman's social, physical, and psychological well-being, leading to embarrassment, low self-esteem, and negative effects on the productivity of working women.

G. Vignoli, *Urodynamics for Urogynecologists*,
https://doi.org/10.1007/978-3-319-74005-8_7

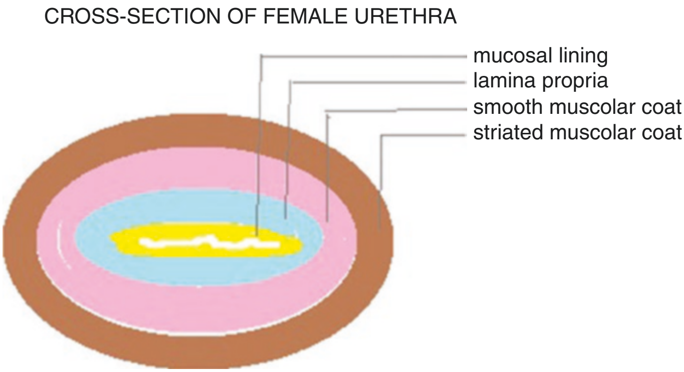


FIGURE 7.1 Structure of female urethra. The female urethra is composed of four separate tissue layers that keep it closed. The inner mucosal lining keeps the urothelium moist and the urethra supple. The vascular spongy coat of lamina propria has an important role in the mucosal seal mechanism. Compression from the middle smooth muscular coat, inner longitudinal and outer circular, helps to maintain the resting urethral closure mechanism. The outer striated seromuscular layer augments, during sudden increase of abdominal pressure, the closure pressure provided by the smooth muscular layer

Stress incontinence is defined by ICS as an involuntary leakage of urine that occurs with increases in intra-abdominal pressure (e.g., with exertion, sneezing, coughing, laughing) in the absence of a bladder contraction. Stress incontinence is the most common type of incontinence in younger women, with the highest incidence between 45 and 49 years.

Anatomically the female urethra (Fig. 7.1) is a multilayered tube, containing mucosa, smooth and striated muscle, and vascular and nerve tissue and is integrated with its surroundings, i.e., striated pelvic floor muscles and connective tissue including the endopelvic fascia. Anatomical abnormalities are relevant in relation to its function.

Mechanisms of stress incontinence include urethral hypermobility and intrinsic sphincteric deficiency (ISD). In most of the patients, the two mechanisms coexist (Fig. 7.2).

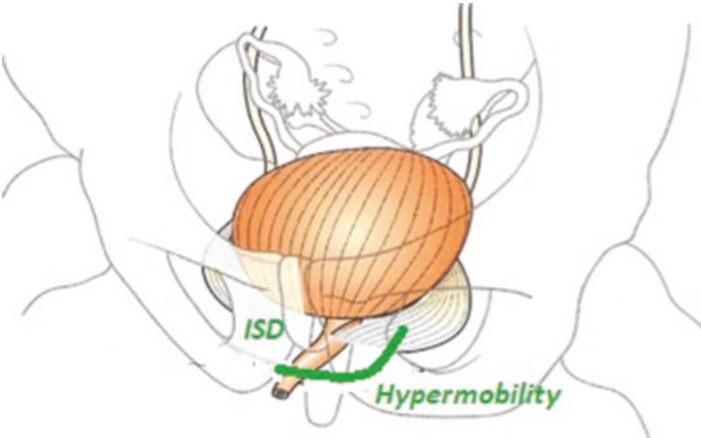


FIGURE 7.2 Mechanisms of stress urinary incontinence

Urethral hypermobility originates from an insufficient support of the urethra and bladder neck by weak pelvic floor musculature and/or poor vaginal connective tissue.

This causes the urethra and bladder neck to lose the ability to completely close against the anterior vaginal wall with the increases in intra-abdominal pressure (e.g., from coughing or sneezing) leading to incontinence. Insufficient urethral support may be related to loss of connective tissue and/or muscular strength due to chronic pressure (i.e., high-impact activity, chronic cough, or obesity) or trauma due to childbirth, particularly vaginal deliveries. Mid-urethral slings and traditional urethropexies are aimed at providing a backboard of support for the urethra.

Intrinsic sphincteric deficiency (ISD) is another form of stress urinary incontinence (SUI) that results from a loss of urethral tone that normally keeps the urethra closed. This can occur in the presence or absence of urethral hypermobility and typically results in severe urinary leakage even with minimal increases in abdominal pressure. In general, ISD results from neuromuscular damage and can be seen in women who have had multiple pelvic or incontinence surgeries. In general it is a challenge to treat women with ISD, since they have worse surgical outcomes.

A third mechanism, still poorly understood, is that of "urethral instability." Urethral instability is the urodynamic observation of urethral pressure variations in patients with symptoms of LUTD, apart from detrusor overactivity. Interstitial cells (ICs) in the urethra have, in analogy with these in the intestine, i.e., the interstitial cells of Cajal (ICC), been proposed as specialized pacemakers that are involved in the generation of urethral tone. Failure to maintain sufficient and continuous urethral muscle contraction may be a specific pathophysiological entity.

Urgency incontinence is defined by ICS the complaint of involuntary loss of urine associated with urgency, where urgency is defined as a sudden, compelling desire to void which is difficult to defer.

The amount of leakage ranges from a few drops to completely soaked undergarments. Urgency incontinence is more common in older women and may be associated with comorbid conditions that occur with age. It is believed to result from detrusor overactivity, leading to uninhibited (involuntary) detrusor muscle contractions during bladder filling. First line of treatment includes lifestyle modifications, timed micturitions, pelvic floor muscles exercises, and topical estrogens for postmenopausal women. Second line of treatment includes pharmacotherapy with antimuscarinics and mirabegron and percutaneous tibial nerve stimulation. For refractory cases, third line of treatment includes botulinum toxin and sacral neuromodulation.

Mixed urinary incontinence is a condition of both stress and urgency urinary incontinence, is prevalent in 20–36% of women, and is challenging to diagnose and treat because urinary symptoms are variable and guidelines for treatment are not clear. The role of surgery in the treatment of mixed incontinence is controversial because there is thought to be a high failure rate, related in part to preexisting symptomatic or asymptomatic DO. However, there is some evidence that surgery may relieve symptoms of DO, especially if stress symptoms prevail over urge symptoms. Because high-quality evidence is lacking regarding the treatment of mixed urinary

incontinence, treatment generally begins with conservative management emphasizing the most bothersome component. In addition, it has been also hypothesized that transobturator tapes could be preferred in women with MUI compared to retropubic tension-free vaginal tapes due to the more horizontal position of the tape and, consequently, less obstructive mechanism.

7.3 Evaluation of Patients with SUI

Identifying predictors for treatment failure would facilitate improved counseling for women considering surgery to manage their SUI and potentially allow surgeons to modify their approach to improve the outcome for individual patients.

Unfortunately, a standardized process to evaluate patients complaining of SUI is currently lacking; however, specialized societies and health organizations (ICI, AUA/SUFU, EAU, NICE) have published specific recommendations.

The initial goal of the evaluation is to determine whether the patient has uncomplicated SUI (index patient) or complicated SUI (non-index patient).

Note: The index patient is that of an otherwise healthy female with stress urinary incontinence with or without pelvic organ prolapse.

Uncomplicated SUI (index patient) should include the following features:

- Predominant SUI symptom
- A history without having extensive pelvic or anti-incontinence surgery
- Absence of POP beyond the hymen
- Confirmation of incontinence under stress (usually cough)
- Urethral hypermobility
- Absence of a UTI
- PVR (≤150 mL)

Conversely *complicated SUI (non-index patient)* is designated by:

- Additional storage and voiding symptoms
- Recurrent UTI
- Poorly controlled diabetes mellitus
- Neurological disease affecting the lower urinary tract
- Prolapse beyond the hymen
- Absence of urethral hypermobility (<30° deflection)
- PVR ≥ 150 mL

The two opposing components of the evaluation process include, on the one hand, a simple office assessment and, on the other, the invasive urodynamic testing.

7.4 Office Evaluation of Female Incontinence

Office evaluation should be urodynamically oriented. If the symptoms and signs are interpreted in the context of functional urodynamic information, then it may be possible to produce a provisional urodynamic diagnosis, and the following invasive investigations may be omitted or used to test the clinical hypothesis.

According to ACOG and AUGS, basic office evaluation should include six steps:

- History
- Urinalysis
- Physical examination
- Demonstration of stress incontinence
- Assessment of urethral mobility
- Measurement of post-void residual urine

7.4.1 History

The statement "bladder is an unreliable witness" was made by Bates in 1970 in one of the early papers on urodynamics. Despite the shortcomings of the patient's symptoms on

Box 7.1: Examples of Validated Urinary Incontinence Questionnaires

Urogenital Distress Inventory (UDI)
Incontinence Impact Questionnaire (IIQ)
Questionnaire for Urinary Incontinence Diagnosis (QUID)
Incontinence-Quality of Life Questionnaire (I-QoL)
Incontinence Severity Index (ISI)
International Consultation on Incontinence Questionnaire (ICIQ)

diagnosis, they are important and should be assessed in a systematic way, analyzing both filling and voiding phases. Storage symptoms include frequency, nocturia, and urgency. Emptying symptoms include hesitancy, slow stream, intermittency, straining to void, position-dependent micturition, feeling of incomplete emptying, and dysuria.

Views on both symptoms severity and QoL might differ between patients and clinicians. Thus, it is important to include reports of QoL and symptom severity directly from the patient. These reports can be collected in a standardized manner with validated self-assessment questionnaires (see Box 7.1).

Patients with uncomplicated SUI will have classic symptoms of leakage on effort or physical exertion. In contrast, inability to reach the toilet that is associated with urgency indicates the presence of urge UI.

7.4.2 Urinalysis

Urinary tract infections should be identified and treated before initiating further investigation or therapeutic intervention for UI. A urine dipstick assessment should be performed to exclude a UTI and a specimen sent for microscopy and culture if abnormal. If the urinalysis result is negative, the patient's condition is still consistent with uncomplicated SUI.

7.4.3 Physical Examination

Physical examination should include abdominal, vaginal, and neuro-urologic assessment (see Chap. 2) and, specifically, in incontinent female should include the demonstration of stress incontinence and the assessment of urethral hypermobility.

7.4.4 Demonstration of Stress Incontinence

Visualization of fluid loss from the urethra simultaneous with a cough is diagnostic of SUI. Conversely, delayed fluid loss suggests a cough-induced detrusor overactivity.

The cough stress test can be performed with the patient in the supine position during the physical examination. However, if urine leakage is not observed, the cough stress test needs to be repeated with the patient standing and with a full bladder to maximize test sensitivity.

Note: To perform the cough stress test in the standing position, the patient stands near the examination table with 1 ft on the table step, whereas the examiner keeps the labia separated to observe the urethral meatus. If the stress test results negative despite the patient symptoms of SUI, then multichannel UDS is recommended.

7.4.5 Assessment of Urethral Mobility

Anti-incontinence surgery is more successful in women with *urethral hypermobility*, defined as a 30° or greater displacement from the horizontal plane when the patient is straining in a supine lithotomy position. Lack of urethral hypermobility is associated with an increase in the failure rate of mid-urethral sling. Patients who lack urethral hypermobility may be better candidates for urethral bulking agents or retropubic

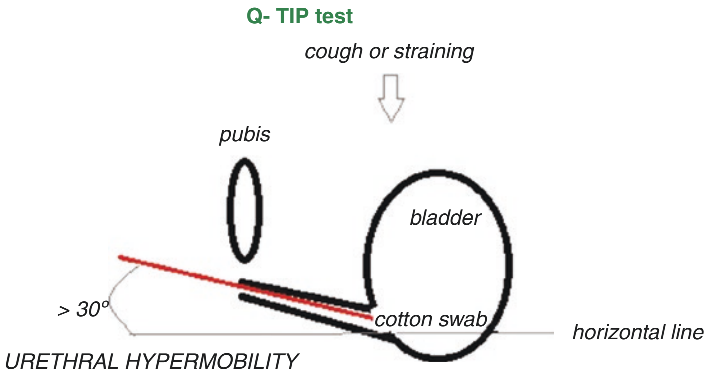


FIGURE 7.3 *Q-tip test* determines the descent of the normal urethrovesical junction contributing to stress incontinence in women. More than 30-degree increase during exertional activities (cough, Valsalva) indicates a hypermobile urethrovesical junction

tape (more obstructive) rather than a transobturator tape (less obstructive).

Q-tip test with cotton swab has been the traditional assessment of urethral mobility (Fig. 7.3).

Bladder neck descent can be also determined by measuring the vertical distance between infero-posterior margin of the symphysis pubis and the bladder neck, at rest, and maximal Valsalva maneuver via perineal ultrasound.

7.4.6 *Post-void Residual Urine (PVR)*

The presence of a post-void residual urine greater than 150 mL can indicate a bladder-emptying abnormality or incontinence associated with chronic urinary retention (previously referred to as overflow incontinence). An elevated post-void residual urine volume in the absence of POP is uncommon and should lead an evaluation of the bladder-emptying mechanism, usually with a pressure-flow study.

Note: Basic office evaluation may be implemented by some supplementary test for a better assessment of the patient. These include voiding diary, pad test, and, when available, uroflowmetry.

7.5 Invasive Urodynamic Testing

The aim of invasive urodynamic testing, apart from confirming SUI, is to differentiate between types of incontinence so that the most effective method of treatment can be selected. To do that, detrusor function is assessed during filling and voiding phases, and intrinsic urethral function is evaluated during filling phase (see Box 7.2).

7.5.1 Confirm SUI

Traditionally, cystometry with cough tests at representative volumes (150 mL) has been the core test for the urodynamic diagnosis of SUI. Urodynamic stress incontinence is the involuntary leakage of urine during increased abdominal pressure in the absence of a detrusor contraction (Fig. 7.4).

7.5.2 Assess Detrusor Activity During Filling

Given that patients undergoing stress incontinence surgery expect not only the resolution of urine leakage but also urgency and frequency symptoms and given that these symptoms are among those that baseline predict postsurgery

Box 7.2: Goals of UDS in SUI

- Confirm SUI
- Assess detrusor activity during filling
- Assess detrusor contractility during voiding
- Assess intrinsic urethral function

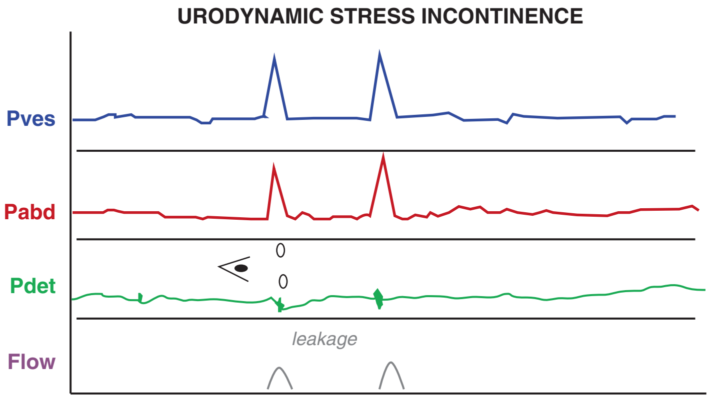


FIGURE 7.4 Urodynamic stress incontinence. Involuntary leakage of urine during increased abdominal pressure in the absence of a detrusor contraction. The patient is usually asked to give sets of strong coughs, and leakage can be assessed by inspection of urethral meatus or recorded through the flowmeter

dissatisfaction, the assessment of detrusor activity may be extremely useful.

7.5.3 *Assess Detrusor Contractility During Voiding*

Unlike OAB, underactive bladder in women is a novel concept, and detrusor underactivity is a difficult to define urodynamic finding (see Chap. 9).

7.5.4 *Assess Intrinsic Urethral Function*

Since combined hypermobility and ISD occurs in the vast majority of incontinent patients, the challenge is to determine the position of a given patient within the spectrum since it may influence the treatment outcome. ISD is commonly associated with more severe symptoms and often is associated with failed previous surgery. In addition to clinical features, most studies base the diagnosis of ISD upon urodynamic appearances using

recognized criteria like Valsalva leak point pressure <60 cm H_2O or a maximum urethral closure pressure <20 cm H_2O). However, the uncertainty over this categorization is recognized by the ICS, which has called for further research in this area. Ranges of surgical treatments, from bulking agents to artificial sphincter, can be used in ISD. A general agreement exists that retropubic slings are more efficacious than transobturator slings. It is acknowledge, however, a need for larger randomized trials, investigating the role of urethral function parameters with relation to effective surgical management of incontinence before reaching definitive conclusions.

7.6 The Debate

The role of urodynamic testing prior to surgery of stress urinary incontinence is under constant debate.

There is lack of universal consensus regarding its value. Whereas some studies support using it, others have failed to demonstrate its routine utility. Detrusor overactivity is the mostly emphasized aspect, but also ISD and detrusor underactivity may play a significant role.

7.6.1 The Case Against

- **NICE guidelines (2006)**

  UDS in women with "pure SUI" is no longer recommended. UDS is indicated only in women with urinary incontinence when there is a clinical suspicion of DO or in patients who have undergone surgery for SUI or anterior compartment prolapse or in women with symptoms suggestive of voiding dysfunction.

- **ValUE (Value of Urodynamic Evaluation) trial (2009)**

  A multicentric US study purely focused on UDS. Women meeting criteria for an anti-incontinence procedure were randomized to preoperative UDS or a simple office-based evaluation. One of the outcomes was to determine if preoperative UDS findings in women with stress predominant incontinence

would affect surgical outcome, with secondary outcomes being cost and utility of performing UDS. At 12 months, no significant difference was seen between the urodynamic testing group and the evaluation-only group with respect to treatment success (76.9% and 77.2%, respectively). The report concluded that UDS did not improve the rate of treatment success and was not necessary in place of a well-performed office-based evaluation (including demonstration of SUI) in women with uncomplicated SUI.

Note: The ValUE trial found that urodynamic studies rarely changed the primary diagnosis of SUI (<1%) but frequently changed the listing of secondary diagnoses of OAB-wet, OAB-dry, voiding dysfunction, or suspected intrinsic sphincter deficiency in over half of women undergoing UDS. Surprisingly, however, these changes of secondary diagnoses rarely changed treatment plans, and therefore, no treatment benefit was realized by the additional UDS.

- **TOMUS (Trial Of Mid-Urethral Slings) trial, 2010** *(ISD do not influence the outcome of sling surgery)*

Preoperative UDS was performed before randomization into a retropubic mid-urethral sling (RMUS) or a transobturator mid-urethral sling (TMUS) and repeated at 12 months post-surgery. Patients and surgeons were blinded to the findings of the preoperative UDS so that they would not be influenced in their treatment decision. The study supported the fact that VLPP did not impact surgical outcome.

- **VUSIS (Value of Urodynamics prior to Stress Incontinence Surgery) I and II** *(different recruitment process)* **trials (2013)**

This multicentric Dutch RCT was examining the role of UDS in women with symptoms of SUI who had UDS findings discordant from history and physical findings and where then randomized to receive either mid-urethral sling or "individual treatment." Individual treatment can include pessary, medical treatment, physiotherapy, or surgery at the discretion of the provider.

Despite the relatively small number of patients (the trial was stopped prematurely because of slow recruitment.), the omission of urodynamics was not inferior to the use of urodynamics in the preoperative workup of women with SUI. Women

with SUI undergoing urodynamics had the risk of a choice for more prudent treatment (other than surgery), which seemed to result in a delay until effective treatment (surgery).

Note: Both ValUE and VUSIS I and II trials are non-inferiority designed trials. A non-inferiority trial is a clinical trial whose objective is to establish a new approach/treatment that is not clinically worse than the active comparison approach/treatment by more than a small predetermined margin.

Example: *if office evaluation of SUI cost 200 Euro and UDS testing 600 Euro and the difference between them is clinically negligible, then my patient can use the less expensive alternative.*

7.6.2 The Case Pros

- **Hermieu** *from the Association Francaise d'Urologie* **(2007)**

In the presence of pure stress urinary incontinence with no other associated clinical symptoms, a complete urodynamic assessment is not mandatory but can be helpful to define the prognosis and inform the patient about her vesicosphincteric function. On the other hand, a complete urodynamic assessment is recommended to investigate complex or complicated urinary incontinence.

- **Agur et al. (2009)**

Only a very small proportion of patients (5.2%) referred with urinary incontinence could be diagnosed as "uncomplicated stress-predominant urinary incontinence" on the basis of symptoms and the sign of urinary loss during stress testing. Furthermore 26.1% of women with a "clearly defined clinical diagnosis" of pure SUI may have important alternative/associated diagnoses that could affect the outcome of surgery for UI. Clinical criteria do not ensure that all women with potentially important urodynamic findings are evaluated appropriately.

- **Digesù et al. (2009)**

Even patients with a history of "uncomplicated" SUI may take advantage from urodynamics since as many as 20% of

them might not need surgery as the first line of treatment due to presence of DO and voiding dysfunction.

- **Guerrette and Davila (2008)**

Mid-urethral sling (Monarch) should be used with caution in women with impaired urethral function.

The maximum urethral closure pressure (MUCP) had a median of 20 cm H_2O in the failures and 45 cm H_2O in the successful patients ($p > 0.001$).

The median VLPP at cystometric capacity (VLPPcap) in the failures was 32 cm H_2O compared to 71 cm H_2O in the successes ($p > 0.001$).

- **Serati et al. (2013)**

UDS is able to show that several patients (approx. 20%) with symptoms of pure SUI present an underlying DO and do not require surgery, even 1 year after UDS. In these patients, antimuscarinic treatment appears to ensure a good rate of cure; thus, UDS could lead to the avoidance of several surgical procedures.

- **Finazzi-Agrò et al.** *from the Società Italiana di Urodinamica* **(2013)**

"Uncomplicated" patients represent a minority of patients evaluated before surgery for female SUI. According to the data of six referral centers in Italy, the role of urodynamics in female incontinence has not been challenged yet, and the investigation seems still mandatory.

7.6.3 The Future

- **INVESTIGATE-I** (INVasive Evaluation before Surgical Treatment of Incontinence Gives Added Therapeutic Effect?)

A mixed-methods study comprising a pragmatic multicenter randomized pilot trial, economic evaluation, survey of clinicians' views about invasive urodynamic testing, and qualitative interviews with clinicians and trial participants.

The study should be a definitive randomized controlled trial of invasive urodynamic testing compared with basic clinical assessment and noninvasive testing in women potentially suitable for surgical treatment for urinary incontinence.

Key Notes for Clinical Practice

- Patients with stress urinary incontinence desiring to undergo surgical treatment should be divided in:

Index patient (uncomplicated SUI)	**Non-index patient (complicated SUI)**
History of SUI or MUI with prevalent SUI	Urgency-predominant SUI
Objective demonstration of SUI with full bladder	Inability to demonstrate SUI
Absent post-void residual urine	Elevated post-void residual urine
Negative urinalysis	Positive urinalysis or hematuria
	POP-Q stage 3 or higher
	Failure of prior surgery
	Known or suspected NLUTD

- Physicians may omit urodynamics testing for the index patient.
- Physicians should perform urodynamic testing for the non-index patient.
- Despite urodynamic findings, physicians should counsel the patients on potential complications of surgery.

(Modified from Kobashi K, Albo M, Dmochowsky R, Ginsberg DA, Goldman HB, Gomelsky A, et al. Surgical treatment of female stress urinary incontinence. AUA/SUFU Guidelines, 2017)

Note: Despite the clear indications of the guidelines, a recent survey reported the 51% of urogynecologsts would perform UDS before surgical correction of uncomplicated SUI, while 79% would perform UDS only if no urine leakage can be demonstrated on physical examination. Furthermore, 87% of clinicians would perform UDS before surgical correction of SUI in patients with mixed urinary incontinence.

Prior to surgical repair of POP, 27% of urogynecologists would only perform a physical examination to rule out SUI, while 34% would perform UDS as well.

Suggested Readings

Overviews

Serati M, Ghezzi F. The epidemiology of urinary incontinence: a case still open. Ann Transl Med. 2016;4:123.

Hannestad YS, Rortveit G, Sandvik H, Hunskaar S. A community-based epidemiological survey of female urinary incontinence: the Norwegian EPINCONT study. Epidemiology of incontinence in the County of Nord-Trøndelag. J Clin Epidemiol. 2000;53:1150–7.

Ebbesen MH, Hunskaar S, Rortveit G, Hannestad YS. Prevalence, incidence and remission of urinary incontinence in women: longitudinal data from the Norwegian HUNT study (EPINCONT). BMC Urol. 2013;13:27.

McGuire EJ. Urodynamic findings in patients after failure of stress incontinence operations. Prog Clin Biol Res. 1981;78:351–60.

Chaikin DC, Rosenthal J, Blaivas JG. Pubovaginal fascial sling for all types of stress urinary incontinence: long-term analysis. J Urol. 1998;160:1312–6.

Ghoniem G, Hammett J. Female pelvic medicine and reconstructive surgery practice patterns: IUGA member survey. Int Urogyn J. 2015;26(10):1489–94.

Park K, Kim S, Huh J-S, Kim Y-J. A study of clinical predictors associated with intrinsic sphincter deficiency in women with stress urinary incontinence. Int Neurourol J. 2017;21:139–42.

Kirschner-Hermanns R, Anding R, Rosier P, Birder L, Andersson KE, Djurhuus JC. Fundamentals and clinical perspective of urethral sphincter instability as a contributing factor in patients with lower urinary tract dysfunction—ICI-RS 2014. Neurourol Urodyn. 2016;35:318–23.

Komesu YM, Schrader RM, Ketai LH, Rogers RG, Dunivan GC. Epidemiology of mixed, stress, and urgency urinary incontinence in middle-aged/older women: the importance of incontinence history. Int Urogynecol J. 2016;27:763–72.

Malek JM, Ellington DR, Jauk V, Szychowski JM, Parden AM, Richter HE. The effect of age on stress and urgency urinary incontinence outcomes in women undergoing primary midurethral sling. Int Urogynecol J. 2015;26:831–5.

Myers DL. Female mixed urinary incontinence: a clinical review. JAMA. 2014;311:2007–14.

The Debate: Cons

NICE. National Institute for Health and Care Excellence Urinary incontinence: the management of urinary incontinence in women. NICE clinical guideline, vol 40. London: National Institute for Health and Care Excellence; 2006. p. 1–36.

Nager CW, Brubaker L, Daneshgari F, Litman HG, Dandreo KJ, Sirls L, et al. Design of the value of urodynamic evaluation (ValUE) trial: a non-inferiority randomized trial of preoperative urodynamic investigations. Contemp Clin Trials. 2009;30:531–9.

Nager CW, Brubaker L, Litman HJ, Zyczynski HM, Varner RE, Amundsen C, et al. A randomized trial of urodynamic testing before stres sincontinence surgery. N Engl J Med. 2012;366:1987–97.

Richter H, Albo M, Zyczynsky H, Kenton K, Norton PA, Sirls LT, et al. Retropubic versus transobturator midurethral slings for stress incontinence. New Engl J Med. 2010;362:2066–76.

Rachaneni S, Latthe P. Does preoperative urodynamics improve outcomes for women undergoing surgery for stress urinary incontinence? A systematic review and meta-analysis. BJOG. 2015a;122:8–16.

Clement KD, Lapitan MC, Omar MI, Glazener CM. Urodynamic studies for management of urinary incontinence in children and adults. Cochrane Database Syst Rev. 2013;29:CD003195.

Glazener CM, Lapitan MC. Urodynamic investigations for management of urinary incontinence in adults. Cochrane Database Syst Rev. 2002;3:CD003195.

van Leijsen SA, Kluivers KB, Mol BW, Broekhuis SR, Milani AL, Bongers MY, et al. Can preoperative urodynamic investigation be omitted in women with stress urinary incontinence? A non-inferiority randomized controlled trial. Neurourol Urodyn. 2012;31:1118–23.

van Leijsen SA, Kluivers KB, Mol BW, Hout Ji, Milani AL, Roovers JP, et al. Value of urodynamics before stress urinary incontinence surgery: a randomized controlled trial. Obstet Gynecol. 2013;121:999–1008.

van Leijsen SA, Mengerink BB, Kluivers KB. Urodynamics before stress urinary incontinence surgery. Curr Opin Obstet Gynecol. 2014;26:398–403.

Zimmern P, Litman H, Nager C, Sirls L, Kraus SR, Kenton K, et al. Pre-operative urodynamics in women with stress urinary incontinence increases physician confidence, but does not improve outcomes. Neurourol Urodyn. 2014;33:302–6.

Rachaneni S, Latthe P. Does preoperative urodynamics improve outcomes for women undergoing surgery for stress urinary incontinence? A systematic review and meta-analysis. BJOG. 2015b;122:8–16.

Norton P, Nager C, Brubaker L, Lemack GE, Sirls LT, Holley R, et al. The cost of preoperative urodynamics: a secondary analysis of the ValUE trial. Neurourol Urodyn. 2016;35:81–4.

Suneetha R, Pallavi L. Urodynamics before surgery for stress urinary incontinence. Eur Urol Focus. 2016;2:275–6.

Medina C, Costantini E, Petri E, Mourad S, Singla A, Rodriguez-Colorado S, et al. Evaluation and surgery for stress urinary incontinence: a FIGO working group report. Neurourol Urodyn. 2017;36:518–28.

The Debate: Pro

Hermieu JF, Comitéd'Urologie et de Pelvi-périnéologie de la Femme Association Francaise d'Urologie. Recommendations for the urodynamic examination in the investigation of non-neurological female urinary incontinence. Prog Urol. 2007;17(6 Suppl 2):1264–84.

Agur W, Housami F, Drake M, Abrams P. Could the National Institute for Health and Clinical Excellence guidelines on urodynamics in urinary incontinence put some women at risk of a bad outcome from stress incontinence surgery? BJU Int. 2009;103:635–9.

Digesu GA, Hendricken C, Fernando R, Khullar V. Do women with pure stress urinary incontinence need urodynamics? Urology. 2009;74:278–81.

Guerrette N, Davila B. Transobturator slings for stress incontinence: using urodynamic parameters to predict outcomes. Int Urogynecol J. 2008;19:97–102.

Serati M, Cattoni E, Siesto G, Braga A, Sorice P, Cantaluppi S, et al. Urodynamic evaluation: can it prevent the need for surgical intervention in women with apparent pure stress urinary incontinence? BJU Int. 2013;112:E344–50.

Dillon BE, Zimmern PE. When are urodynamics indicated in patients with stress urinary incontinence? Curr Urol Rep. 2012;13:379–84.

Giarenis I, Cardozo L. What is the value of urodynamic studies before stress incontinence surgery? BJOG. 2013;120:130–2.

Finazzi-Agrò E, Serati M, Salvatore S, Del Popolo G. Comments on "A randomized trial of urodynamic testing before stress-incontinence surgery" (N Engl J Med. 2012 May 24;366(21):1987-1997) From the Italian Society of Urodynamics. Neurourol Urodyn. 2013;32:301–2.

Topazio L, Frey J, Iacovelli V, Perugia C, Vespasiani G, Finazzi-Agrò E. Prevalence of "complicated" stress urinary incontinence in female patients: can urodynamics provide more information in such patients? Int Urogynecol J. 2015;26:1333–9.

Serati M, Finazzi-Agrò M. Urodynamics before surgery for stress urinary incontinence: the urodynamic examination is still one of the best friends of the surgeon and of patients with stress urinary incontinence. Eur Urol Focus. 2016;2:271–3.

Serati M, Topazio L, Bogani G, Costantini E, Pietropaolo A, Palleschi G, et al. Urodynamics useless before surgery for female stress urinary incontinence: are you sure? Results from a multicenter single nation database. Neurourol Urodyn. 2016;35:809–12.

Future Research

Hilton P, Armstrong N, Brennand C, Howel D, Shen J, Bryant A, et al. A mixed methods study to assess the feasibility of a randomised controlled trial of invasive urodynamic testing versus clinical assessment and non-invasive tests prior to surgery for stress urinary incontinence in women: the INVESTIGATE-I study. Trials. 2015;16:400.

Chapter 8
UDS in Pelvic Organ Prolapse Syndrome (POP-S)

8.1 Background

Pelvic organ prolapse (POP) is the herniation of the pelvic organs to or beyond the vaginal walls. It is an age-dependent problem which has been reported to affect 50% of the parous women causing a variety of pelvic, urinary, bowel, and sexual symptoms. Up to 20% of women will require at least one surgery for correction of POP in their lifetime, with an estimated 30% reoperation rate.

POP is categorized according to the affected compartment:

- Anterior (cystocele)
- Posterior (rectocele)
- Apical (descent of the uterus or bowel—enterocele—following hysterectomy)

The presence of associated symptoms is very important since treatment of urinary or fecal symptoms is typically coordinated with treatment for POP. Asymptomatic POP may not require treatment.

Note: Women may not be aware of a vaginal prolapse until it reaches the hymen. It follows that many women can be categorized as having prolapse on physical examination, although they do not manifest any symptoms consistent with

G. Vignoli, *Urodynamics for Urogynecologists*,
https://doi.org/10.1007/978-3-319-74005-8_8

prolapse. Terms such as "subclinical" or "preclinical" prolapse have been suggested, in order to distinguish asymptomatic women from those with symptoms of prolapse. It is not known whether women with subclinical prolapse are at higher risk for the development of clinically evident prolapse. However, there are no data that supports intervention to promote prevention of possible future pelvic floor disorders.

8.2 Classification of Pelvic Organ Prolapse

The large number of different grading systems that have been used is reflective of the difficulty in designing an objective, reproducible system of grading prolapse. Intra- and interobserver variability is often important and may lead to confusion. This makes it difficult to compare successive examinations over time in the same woman or between different women.

Evaluation for POP can be done using one of several clinical quantification systems. The Baden Walker classification (1971) was one of the first classification systems used. The POP-Q system introduced in 1996 has become the internationally accepted standard for reporting POP in research today. POP-Q system includes nine measurements taken in centimeters from fixed points. The six points are measured to evaluate anterior, posterior, and apical descent of pelvic organs in relation to a fixed point of reference—the hymen—at maximum Valsalva. Additionally, measurements were taken of the genital hiatus, perineal body, and total vaginal length. The POP-Q system has not been widely adopted in clinical practice particularly by non-urogynecologist, owing somewhat to difficulty in learning the assessment. Because of the complexity of the POP-Q, a more simplified version (S-POP-Q) has been developed by Swift in 2002 and subsequently adopted by the IUGA. Because of its simplicity, the S-POP-Q system is now recommended for routine clinical practice over the more complex standard POP-Q system.

8.2.1 S-POP-Q: Practical Tips

For the S-POP-Q, the four areas examined include the anterior and posterior vaginal walls, the apex, and the cervix (Fig. 8.1). If the patient underwent hysterectomy, then only three measurements are taken: the anterior and posterior vaginal walls and the cuff. No measuring devices are required for the S-POP-Q, and the investigators have to use rough estimation for identifying those points on the anterior and posterior vaginal segments that are used to represent the respective walls. For examination of the anterior vaginal segment, the speculum is placed into the vagina, and the posterior vaginal wall is retracted to allow for full visualization of the anterior vaginal wall. A point approximately halfway up the anterior vaginal wall or approximately 3 cm proximal to the urethral meatus or hymenal plane is identified. The

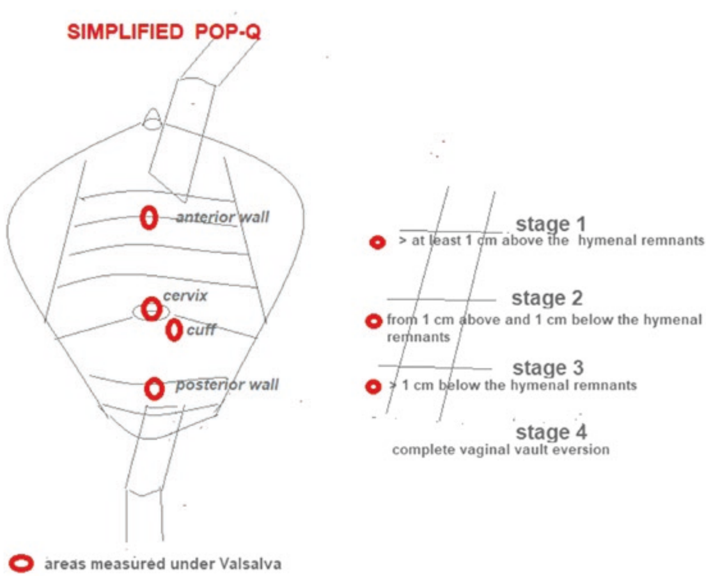


FIGURE 8.1 S-POP-Q landmarks and relationship between measurements and POP stage

subject is then instructed to Valsalva or cough in a forceful fashion and where that point descend in relation to the hymenal plane is recorded as the suitable stage of the anterior vaginal wall. The posterior segment is examined in a similar fashion. The cervix is evaluated by placing a Sims speculum in the vagina and directly observing its descent during a Valsalva or cough to determine its stage in relation to the hymenal plane.

The staging system for each segment is:

- Stage 1: Prolapse where the given point remains at least 1 cm above of the hymenal remnants.
- Stage 2: Prolapse where the given point descends to the introitus, defined as an area extending from 1 cm above to 1 cm below the hymenal remnants.
- Stage 3: Prolapse where the given point descends greater than 1 cm past the hymenal remnants, but does not represent complete vaginal vault eversion or complete procidentia uteri. This implies that at least some portion of the vaginal mucosa is not everted.
- Stage 4: Complete vaginal vault eversion or complete procidentia uteri. This implies that the vagina and/or uterus is maximally prolapsed with essentially the entire extent of the vaginal mucosa everted.

Note: With the introduction of MRI, prolapse description uses a nomenclature based on a different axis to clinical examination (i.e., sagittal section). The discrepancy between MRI and POP-Q is significant. However, the concept of the H and M lines, while measureable and reproducible, has not demonstrated any advantage over clinical examination. Until we have a clinically relevant staging system, attempting to correlate radiologic findings with clinical findings will prove problematic.

8.3 The Role of Urodynamics Before Prolapse Surgery

The role of urodynamic testing before prolapse surgery is contentious and a hotly debated topic in urogynecology.

Prolapse surgery has to deal with three main functional problems:

- SUI syndrome
- OAB syndrome
- Voiding dysfunction syndrome
- POP and SUI Syndrome

8.4 POP and SUI Syndrome

Urogynecologists are often challenged by the persistence or the de novo development of stress urinary incontinence following POP repair.

About 40% of all patients with genital prolapse report stress incontinence. In about half of the 60% patients that do not report stress incontinence, occult urinary stress incontinence can be detected. In these patients, stress incontinence is masked due to kinking or compression of the urethra by the prolapse (see Box 8.1).

Box 8.1: Incidence of POP and SUI

60% of pts with POP are continent.
40% of pts with POP complain SUI.
20% of continent pts with POP have "occult SUI."
Note: "Occult SUI" is an incontinence observed only after the reduction of a coexistent prolapse.

The combination of prolapse surgery with an anti-incontinence procedure is frequently considered as a suitable option to overcome the problem. In particular, three groups of patients can be considered:

- Women with coexisting SUI
- Women asymptomatic for SUI
- Women asymptomatic for SUI with occult SUI

The advantage of combining prolapse and stress incontinence surgery is that only few patients report stress incontinence following such combination. However, this combination has been associated with an increased risk on complications, of which the development of obstructive micturition symptoms, overactive bladder symptoms, and bladder retention are the most important ones. Furthermore, combining two procedures may be unnecessary as performing only prolapse surgery may cure stress incontinence. Thus, the relative benefit of adding an anti-incontinence procedure as a primary option requires evaluation.

To date, there are three main approaches to this issue:

- Perform an incontinence procedure in *all* patients undergoing POP surgery.
- Perform an incontinence procedure in *no* patients undergoing POP surgery.
- Perform an incontinence procedure in *some* patients undergoing POP surgery (namely, those with symptoms of SUI or those with demonstrable SUI on stress testing or those with occult SUI on stress testing after prolapse reduction).

Note: Currently, there are two main methods to repair POP:

- Vaginally through fascial repair or MESH reinforcement
- Abdominally with sacrocolpopexy (open, laparoscopic, and robotic)

Likewise, in the treatment of SUI, there are two procedures considered to be gold standard because of similar cure rates:

- Vaginally with the introduction of a midurethral sling (MUS)
- Abdominally with a Burch colposuspension

When combination surgery is performed vaginally, repair is often combined with a MUS, whereas sacrocolpopexy is more likely to be combined with a Burch colposuspension.

8.4.1 Incontinence Surgery in all Patients

Evidence to support this approach come from:

- **CARE trial**. Women undergoing abdominal sacrocolpopexy who did not have any significant symptoms of SUI (answers of never or rarely to the SUI questions on the MESA questionnaire) were randomized to either a Burch suspension or no Burch suspension (the control). Postoperatively significantly more women in the no Burch group developed bothersome SUI than in the Burch group (24.5% vs. 6.1%). No significant postoperative difference in the frequency of urge incontinence was observed.
- **OPUS trial**. Women without stress urinary incontinence undergoing vaginal surgery for pelvic organ prolapse (stage 2 or higher) were randomly assigned to receive either a midurethral sling or sham incisions during surgery. At 12 months, urinary incontinence (allowing for subsequent treatment of incontinence) was present in 27.3% and 43.0% of patients in the sling and sham groups. Adverse events including urinary tract infection (31% vs. 18.3%) and incomplete bladder emptying 6 weeks after surgery (3.7% vs. 0%) were higher in the sling group than in the sham group.
- **CUPIDO 1** (*women with incontinence*) **and 2** (*women with occult SUI*) *trials*. Women were randomly assigned to undergo vaginal prolapse repair with or without MUS. Women with POP and coexisting SUI are less likely to have UI after vaginal prolapse repair with MUS compared with prolapse repair only and are less likely to undergo additional treatment for SUI.

8.4.2 Incontinence Surgery in No Patients

At the opposite end of the spectrum, there is the strategy to not perform an incontinence surgery in patients undergoing POP repair whether or not they have symptoms or signs of SUI preoperatively. Patients with bothersome SUI postoperatively will

be candidate for an antincontinence procedure. The clear advantage of this approach is that no patient who does not have SUI postoperatively will have had an unnecessary antincontinence procedure. The disadvantage of course is that many patients will have SUI postoperatively and require secondary surgery.

Evidence to support this approach comes from the observation that 45% of the patients, even with clinical SUI, have resolution of SUI after POP surgery alone. The disadvantages with this approach include the fact that many women—apparently greater than 50%—will require a second supplementary surgery. The situation could be particularly stressful in patients who have an occult SUI for which the management of an "unknown" incontinence, while waiting the second intervention, may be significantly disappointing.

8.4.3 Incontinence Surgery in Some Patients (namely, Patients with Coexisting and Occult SUI-S)

This selective approach is used by the majority of clinicians. According to AUA SUI-S Guidelines, "few would disagree that operations for SUI should be confined to those patients who actually have demonstrable SUI *including occult SUI demonstrable only after reduction of pelvic organ prolapse*."

The advantage of this approach is that very few patients with SUI will be missed, and unnecessary treatment with the related risks will be avoided in most patients who do not need an antincontinence surgery.

In this group of patients, preoperative screening for coexisting and occult SUI seems pivotal.

8.5 Testing for Occult SUI and Assessment of Urethral Function

Occult SUI is defined as an incontinence that is not symptomatic but becomes apparent only during clinical or urodynamic testing when the prolapse is reduced. Occult stress

incontinence is also referred to as latent, hidden, iatrogenic, or potential incontinence.

Signs in medical history that may suggest occult SUI include:

- Incontinence that improved or resolved as prolapse worsened
- The need to manually replace the prolapsed structures into the vagina to void
- Worsening or development of SUI with use of a pessary

8.5.1 Provocative Tests

Prolapse reduction testing may be performed as part of office testing of SUI or during urodynamic evaluation. Both approaches appear to have a similar predictive value for the development of postoperative SUI.

Prolapse reduction may be performed in semilithotomic position using fingers, large cotton swab, single speculum blade, or pessary. The bladder should be filled at a volume between 100 and 300 mL, the latter being more reliable in predicting postoperative SUI.

Pessaries may be less effective at detecting SUI than other methods because they increase the maximum urethral closure pressure and functional urethral length. However, once positioned, the pessary should be left in situ for at least 1 week to see whether continuous use would elicit UI, regardless of any leakage on the objective tests. Care should be taken in positioning the pessary. While elevating the prolapsed structures, it is important to avoid obstructing the urethra and placing the anterior vaginal wall under excessive tension, which could mask incontinence by distorting the pelvic anatomy.

8.5.2 Urodynamic Testing

Invasive UDS provides more information on urethral function than office provocative stress test.

Urodynamic parameters of urethral function include:

- Urodynamic observation of SUI (leakage under cough or Valsalva without any concomitant detrusor contraction)
- VLPP with a cutoff (normal vs. ISD) greater than 60 cm H_2O
- MUCP with a cutoff (normal vs. ISD) greater than 20 cm H_2O
- (Less commonly) % pressure transmission with a cutoff (normal vs. defect in support) greater than 90%

Despite lack of solid evidence, these parameters may be useful in choosing the type of incontinence surgery to be used concurrently with POP repair, according to comparative data on the effectiveness of different types of mid-urethral slings.

Note: The retropubic tape in that is closer to perpendicular to the urethral axis creates greater circumferential compression of the urethra providing a better support.

Figure 8.1 summarizes the risk of SUI after POP repair according to different approaches.

8.6 POP and OAB Syndrome

Since both POP-S and OAB-S are frequently seen in the elderly female population, it would be expected that the two conditions are frequently encountered in the same patient. Up to 96% of women with POP report LUTS with mixed urinary incontinence predominating. If there is a causal relationship, it could be anticipated that OAB symptoms would improve after successful treatment of POP. Unfortunately, this is not the rule, and it is still unclear whether there is a causal relationship between the two conditions. Nevertheless, many women with either urgency or urge incontinence are sent to POP surgery for solving their problems. Persistent OAB symptoms are associated with poor patient satisfaction as the majority of the patients expect complete postoperative resolution of all their LUTS.

8.6.1 Key Facts About POP and OAB Syndrome

- The prevalence of OAB symptoms is greater in the POP patients than in patients of the same age group without POP, accounting for some role of POP in development of OAB symptoms.
- POP can cause bladder outlet obstruction, and this is often regarded as an important mechanism for developing OAB in these patients in a way not dissimilar to that of men with prostatic bladder outlet obstruction. Patients with prolapse have a lower maximum flow rate (Qmax) during voiding and higher PVR than patients without a prolapse. These findings suggest that some degree of obstruction during voiding may play a role in DO concomitant with POP.
- Unfortunately, diagnosis of obstruction is a difficult issue in female (see Chap. 10). Recently, it has been reported that the criterion (**pdetmax > 2Qmax)** is the best determinant of BOO in women (ICI-RS 2014).
- Treatment of POP (by surgery or simple pessary) gives an improvement in OAB complaints as well as DO symptoms in approximately 60% of the patients.
- In the remaining 40% of patients with persistent symptoms after POP repair, pathophysiological mechanisms other than obstruction should be considered, including increased afferent activity from the urothelium and abnormal handling of afferent signals by brain areas, particularly in elderly patients (see Chap. 9).

In support of this view, there are studies indicating a lack of correlation between the prolapsed compartment and the presence of OAB symptoms, when intuitively one would consider an anterior prolapse more at risk of developing OAB symptoms than a central or posterior one. In addition, urgency and urgency incontinence seem to occur more often in women with a less advanced prolapse overall.

- Mechanisms alternative to bladder outlet obstruction should also be applied to 20% of patients who develop de novo urgency urinary incontinence (UUI) after prolapse repair.

Note: The term de novo urgency urinary incontinence is used to describe urgency incontinence that develops following surgical correction of the prolapse and persists after 6 months among women who did not have any urgency preoperative.

In summary, with the current state of our knowledge, there are no means to discern which factors will determine the persistence or disappearance of OAB symptoms after POP repair, and patients should be adequately counseled on that possibility. In any case, prolapse repair should not be offered to women as a primary treatment for detrusor overactivity.

8.7 POP and Voiding Dysfunction Syndrome

A third of women with stage 2 or more POP experience difficulties in emptying the bladder. Voiding difficulties usually disappear postoperatively because the obstruction caused by the prolapse has been corrected. In contrast, voiding dysfunction might develop after surgery because of kinking of the urethra due to the surgical technique. Postoperative voiding dysfunction (including postoperative urinary retention) is a frequent consequence of gynecologic surgery. Its prevalence ranges from 2.5 to 24% after only prolapse surgery reaching the 43% when a concomitant sling placement is undertaken. In most of the patients is a consequence of a failure to relax appropriately the pelvic floor muscles during voiding and is usually temporary. Prolonged retention, lasting 4 weeks or longer after surgery, has a prevalence of 2–4%. In these patients, surgical obstruction should be ruled out through a pressure-flow study in order to decide a sling cut within few weeks. In female complaining voiding difficulties preoperatively, no valid conclusions regarding voiding dysfunction following POP surgery can be drawn from preoperative urodynamic testing due to difficulties in defining and assessing detrusor underactivity in female patents (see Chap. 10).

Key Notes for Clinical Practice

To date, it has not been possible to reach a universal consensus on the role of UDS before prolapse surgery. It is clear that UDS could add some information in women undergoing pelvic organ prolapse surgery and could facilitate counseling of patients. However, there is no evidence that the outcome of surgery is altered by prior UDS.

- *Prolapse + SUI*

In women with prolapse and SUI symptoms, prolapse procedures alone are associated with low success rates for SUI. Concomitant continence procedures (MUS, Burch colposuspension) reduce the risk of postoperative SUI.

- *Prolapse + occult SUI*

In continent women undergoing POP surgery with occult SUI, the addition of continence surgery reduces the rate of postoperative SUI.

- *Prolapse without SUI*

No clear conclusion can be made regarding the management of continent women undergoing prolapse surgery without occult SUI. The risk of incontinence after simple POP repair is relatively low (7%), and the NNT (number need to treat) is probably considerable. To prevent incontinence in one woman, 20 patients should undergo combined surgery, and the benefits of reduced surgery might outweigh the risks of combined surgery.

- *Antincontinence procedure*

A retropubic tape appears to be more suitable than a transobturator tape in women with intrinsic sphincter deficiency, diagnosed using urethral pressure profilometry and/or Valsalva leak point pressures.

- *OAB symptoms*

Preoperative bladder overactivity may resolve in 40% of patients undergoing POP surgery, and de novo bladder overactivity may occur in 20%. No clear conclusions can be drawn from preoperative urodynamic testing, and patients should be counseled about persistent postoperative OAB symptoms.

- *Voiding dysfunction*

Except the evident cases of obstruction, urodynamic testing are not particularly useful in predicting the resolution of voiding symptoms after POP surgery due to the difficulties in assessing detrusor underactivity in female patients.

Overall, when preoperative UDS are suggestive of detrusor overactivity (DO) or detrusor underactivity (DU) and even in patients who habitually void by abdominal straining, an interval rather than a concomitant continence procedure is advisable even in women with occult SUI.

Suggested Readings

POP Classification Systems

Baden WF, Walker TA. Physical diagnosis in the evaluation of vaginal relaxation. Clin Obstet Gynecol. 1972;15:1055–69.

Bump R, Mattiasson A, Bo K, Brubaker L, Delancey JO, Klarskov P, et al. The standardization of terminology of female pelvic organ prolapse and pelvic floor dysfunction. Am J Obstet Gynecol. 1996;175:10–7.

Swift S. Current opinion on the classification and definition of genital tract prolapse. Curr Opin Obstet Gynecol. 2002;14:503–7.

Ghoniem G, Stanford E, Kenton K, Achtari C, Goldberg R, Mascarenhas T, et al. Evaluation and outcome measures in the treatment of female urinary stress incontinence: International Urogynecological Association (IUGA) guidelines for research and clinical practice. Int Urogynecol J. 2008;19:5–33.

Haylen BT, De Ridder D, Freeman RM, Swift SE, Berghmans B, Lee J, et al. An International Urogynecological Association (IUGA)/ International Continence Society (ICS) joint report on the terminology for female pelvic floor dysfunction. Int Urogynecol J. 2010;21:5–26.

Haylen BT, Mahen CHF, Barber MD, Camargo S, Dandolu V, Digesu A, et al. An International Urogynecological Association (IUGA) /International Continence Society (ICS) joint report on the terminology for female pelvic organ prolapse (POP). Int Urogynecol J. 2016;27:165–94.

Overviews

Serati M, Giarenis I, Meschia M, Cardozo L. Role of urodynamics before prolapse surgery. Int Urogynecol J. 2015;26:165–8.

Adelowo A, Dessie S, Rosenblatt PL. The role of preoperative urodynamics in urogynecologic procedures. J Minim Invasive Gynecol. 2014;21:217–22.

Ballert KN. Urodynamics in pelvic organ prolapse when are they helpful and how do we use them? Urol Clin North Am. 2014;41:409–17.

Baessler K, Maher C. Pelvic organ prolapse surgery and bladder function. Int Urogynecol J. 2013;24:1843–52.

Dillon B, Lee D, Lemack G. Urodynamics role in incontinence and prolapse: a urology perspective. Urol Clin North Am. 2012;39:265–72.

Whiteside JL. Making sense of urodynamic studies for women with urinary incontinence and pelvic organ prolapse a urogynecology perspective. Urol Clin North Am. 2012;39:257–63.

Serati M, Salvatore S, Siesto G. Urinary symptoms and urodynamic findings in women with pelvic organ prolapse: is there a correlation? Results of an artificial neural network analysis. Eur Urol. 2011;60:253–60.

Costantini E, Lazzeri M. Urodynamics for pelvic organ prolapse surgery: "par for the course". Eur Urol. 2011;60(2):261.

Roovers JP, van Laar JO, Loffeld C, Bremer GL, Mol BW, Bongers MY. Does urodynamic investigation improve outcome in patients undergoing prolapse surgery? Neurourol Urodyn. 2007;26:170–5.

Romanzi LJ, Chaikin DC, Blaivas JG. The effect of genital prolapse on voiding. J Urol. 1999;161:581–6.

POP and SUI-S

Khullar V, Anding R, Robinson D, Castro-Diaz D, Dmochowski R, Cardozo L. Under what circumstances should stress incontinence surgery be performed at the same time as prolapse surgery? ICI-RS 2015. Neurourol Urodyn. 2017;36:909–14.

Visco AG, Brubaker L, Nygaard I, Richter HE, Cundiff G, Fine P, et al. The role of preoperative urodynamic testing in stress-continent women undergoing sacrocolpopexy: the Colpopexy and Urinary Reduction Efforts (CARE) randomized surgical trial. Int Urogynecol J Pelvic Floor Dysfunct. 2008;19:607–14.

Brubaker L, Nygaard I, Richter HE, Visco A, Weber AM, Cundiff GW, et al. Two-year outcomes after sacrocolpopexy with and without Burch to prevent stress urinary incontinence. Obstet Gynecol. 2008;112:49–55.

Maher C, Feiner B, Baessler K, Adams EJ, Hagen S, Glazener CM. Surgical management of pelvic organ prolapse in women. Cochrane Database Syst Rev. 2010;4:CD004014.

van der Steen A, van der Ploeg M, Dijkgraaf MG, van der Waart H, Roovers JP. Protocol for the CUPIDO trials; multicenter randomized controlled trials to assess the value of combining prolapse surgery and incontinence surgery in patients with genital prolapse and evident stress incontinence (CUPIDO I) and in patients with genital prolapse and occult stress incontinence (CUPIDO II). BMC Womens Health. 2010;10:16.

Wei J. A mid urethral sling prevents incontinence among women undergoing vaginal prolapse repair—the OPUS trial. Neurourol Urodyn. 2011;30:809–10.

van der Ploeg JM, van der Steen A, Oude Rengerink K, van der Vaart CH, Roovers JP. Prolapse surgery with or without stress incontinence surgery for pelvic organ prolapse: a systematic review and meta-analysis of randomised trials. BJOG. 2014;121:537–47.

van der Ploeg JM, Oude Rengerink K, van der Steen A, van Leeuwen JH, Stekelenburg J, Bongers MY, et al. Transvaginal prolapse repair with or without the addition of a midurethral sling in women with genital prolapse and stress urinary incontinence: a randomised trial. BJOG. 2015;122(7):1022–30.

POP and OAB-S

Tran H, Chung DE. Incidence and management of de novo lower urinary tract symptoms after pelvic organ prolapse repair. Curr Urol Rep. 2017;18:87.

Kim MS, Lee GH, Na ED, Jang JH, Kim HC. The association of pelvic organ prolapse severity and improvement in overactive bladder symptoms after surgery for pelvic organ prolapse. Obstet Gynecol Sci. 2016;59:214–9.

Tomoe H. Improvement of overactive bladder symptoms after tension-free vaginal mesh operation in women with pelvic organ prolapse: correlation with preoperative urodynamic findings. Int J Urol. 2015;22:577–80.

Dieter AA, Edenfield AL, Weidner AC, Siddiqui NY. How does site of pelvic organ prolapse repair affect overactive bladder symptoms? Female Pelvic Med Reconstr Surg. 2014;20:203–7.

Basu M, Wise B, Duckett J. Urgency resolution following prolapse surgery: is voiding important? Int Urogynecol J. 2013;24:1309–13.

Maher CM, Feiner B, Baessler K, Glazener CM. Surgical management of pelvic organ prolapse in women: the updated summary version Cochrane review. Int Urogynecol J. 2011;22:1445–57.

Long CY, Hsu CS, Wu MP, Liu CM, Chiang PH, Juan YS, Tsai EM. Predictors of improved overactive bladder symptoms after transvaginal mesh repair for the treatment of pelvic organ prolapse: predictors of improved OAB after POP repair. Int Urogynecol J. 2011;22:535–42.

Foster RT Sr, Barber MD, Parasio MF, Walters MD, Weidner AC, Amundsen CL. A prospective assessment of overactive bladder symptoms in a cohort of elderly women who underwent transvaginal surgery for advanced pelvic organ prolapse. Am J Obstet Gynecol. 2007;197:82e1–4.

POP and VD-S

Zhang L, Zhu L, Liang S, Xu T, Lang J. Short-term effects on voiding function after mesh-related surgical repair of advanced pelvic organ prolapse. Menopause. 2015;22(9):993.

Geller E. Prevention and management of postoperative urinary retention after urogynecologic surgery. Int J Women Health. 2014;6:829–38.

Maher C, Schmid C, Baessler K, Feiner B. Surgical management of pelvic organ prolapse in women. Cochrane Database Syst Rev. 2013;(4):CD004014.

Hakvoort RA, Dijkgraaf MG, Burger MP, Emanuel MH, Roovers JP. Predicting short-term urinary retention after vaginal prolapse surgery. Neurourol Urodyn. 2009;28:225–8.

Fitzgerald MP, Kulkarni N, Fenner D. Postoperative resolution of urinary retention in patients with advanced pelvic organ prolapse. Am J Obstet Gynecol. 2000;183:1361–3.

Chapter 9
UDS in Overactive Bladder Syndrome (OAB-S)

9.1 Background

Overactive bladder syndrome (OAB-S) is a symptom complex whose hallmark is the urinary urgency (the sudden compelling desire to void that is difficult to defer), usually accompanied by frequency and nocturia, and sometimes urgency incontinence in the absence of any urinary tract infection or another obvious pathology.

OAB is further subclassified as “OAB wet” if associated with urinary incontinence or “OAB dry” without incontinence. OAB may be idiopathic (non-neurological) or secondary to a neurological cause (e.g., multiple sclerosis, spinal cord injury). The overall prevalence is approximately 16% in general population. Both men and women demonstrate an age-related increase in the prevalence of OAB; however, this is more pronounced in women, particularly after the age of 40 (Fig. 9.1).

In addition, men are more likely to have OAB dry and females to suffer from OAB wet. OAB has a significant effect on quality of life (QoL), particularly if associated with incontinence.

The definition of OAB is based on symptoms only. Although the symptoms are suggestive of detrusor overactivity, this is not a rule of thumb, since only a proportion of patients have detrusor overactivity on urodynamic testing.

G. Vignoli, *Urodynamics for Urogynecologists*,
https://doi.org/10.1007/978-3-319-74005-8_9

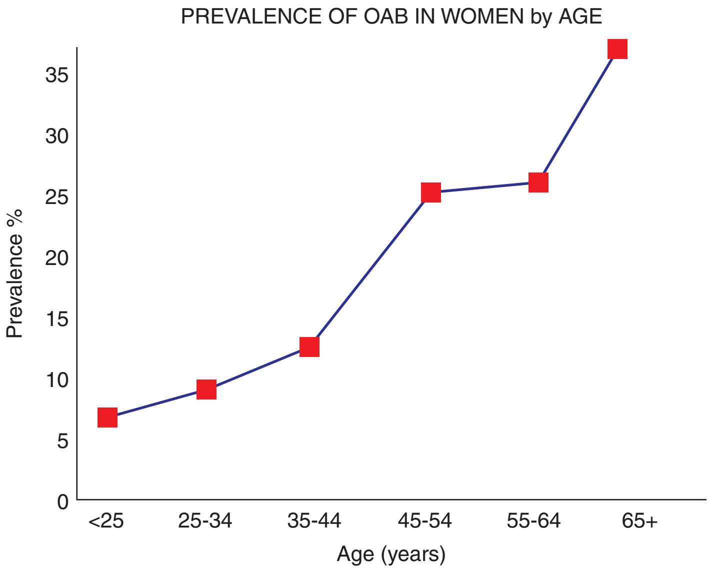


FIGURE 9.1 Prevalence of OAB in women by age

Thus, the terms overactive bladder and detrusor overactivity are distinct, with the latter being a finding in an urodynamic examination.

Historically, urodynamics evaluation has not been recommended in the initial evaluation of OAB, since it is defined primarily by clinical symptoms. Urinalysis and voiding diary are the two most important tools for the initial assessment of the condition. In particular, voiding diary provides quantifiable, objective data that are also useful to analyze the evolution after the prescription of treatment. In postmenopausal women, atrophic vaginitis should be excluded. Assessment of post-void residual (>150 mL) should exclude a chronic urinary retention.

Note: Among noninvasive investigations, urinary NGF has been proposed for the diagnosis of OAB. The neuronal growth factor (NGF) is produced in the urothelium and vesical smooth musculature and modulates the release of neurotransmitters reducing the sensitivity threshold of nociceptive

fibers. Patients with overactive bladder symptoms show higher urinary NGF levels than healthy individuals and achieve lower NGF levels after having been effectively treated. However, an issue in the use of NGF as an overactive bladder biomarker is the fact that it can be elevated in other clinical conditions as well, such as painful bladder syndrome, urinary infections, urinary lithiasis, and bladder tumors.

As the pathophysiology of the OAB syndrome has become more clearly elucidated from recent studies, the role of UDS has again become a topic of discussion as a tool that can provide objective data to reflect these new findings. The utility of UDS in the diagnosis and treatment of OAB is still evolving, but in certain clinical scenarios, especially when empiric treatment has failed, it can provide definitive information that can identify associated pathologies and/or alter the treatment course.

9.2 Current Concepts on Pathophysiology of OAB Syndrome (OAB-S)

OAB-S is a well-defined symptom complex but not very specific for the pathophysiology. The mechanisms behind OAB symptoms remain the subject of debate and ongoing scientific research.

Until the last decade, studies had focused more on the detrusor muscle.

9.2.1 Myogenic Hypothesis

In the myogenic hypothesis, involuntary contractions are caused by changes in myocyte excitability and coupling of myocytes with other myocytes or myofibroblasts. The involuntary contractions that result will provide increased afferent activity and generation of OAB symptoms. Among myogenic mechanisms, great interest has focused in recent years on bladder micromotions. The isolated bladder

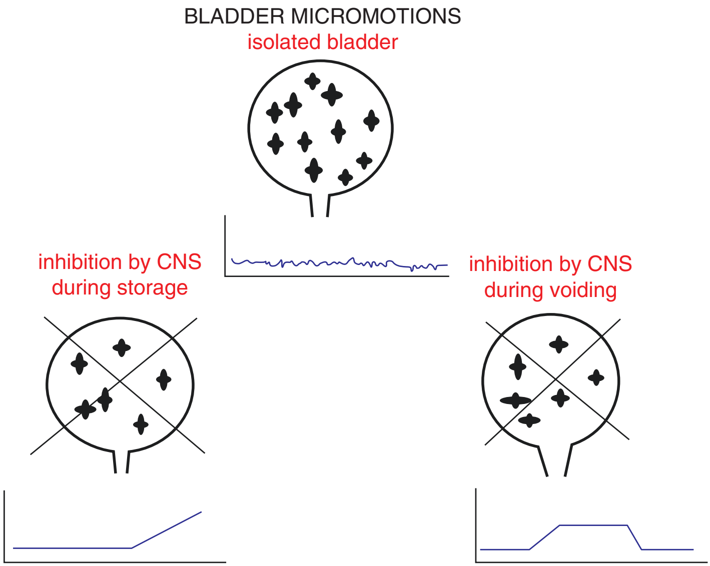


FIGURE 9.2 Bladder micromotions. In vitro, the bladder shows autonomous micromotions, which increase with bladder distension and generate sensory nerve activity. In vivo, micromotions are subordinate to the efferent control of the central nervous system. The loss of efferent inhibition may allow unregulated micromotility with consequent afferent stimulation, which may be responsible for urinary urgency

shows autonomous micromotions, which increase with bladder distension and generate sensory nerve activity (Fig. 9.2).

In vivo, these micromotions are subordinate to the efferent control of the central nervous system, which, in the storage phase, keep the autonomic activity at low level allowing just a signal of a "state of fullness" while maintaining a normal compliance. In OAB, efferent control of the bladder can be impaired, for example, due to peripheral "patchy" denervation. In this case, loss of efferent inhibition may allow an unregulated micromotility, with subsequent afferent stimulation that will predispose to urinary urgency.

Next to myogenic hypothesis, two more underlying mechanisms for OAB symptoms have been suggested by ICS including:

- An increased afferent activity
- An abnormal handling of afferent signals by the brain

9.2.2 Increased Afferent Activity

In the last years, new evidence has highlighted the role of the afferent system and, in particular, emphasized the important afferent role played by the urothelium/suburothelium. The urothelium is an epithelial tissue that lines the urinary tract. It is composed of approximately three to five urothelial cell layers and forms an effective barrier to harmful components in urine. In addition to this barrier function, the urothelium also has sensory and signaling functions, which are currently hot topics in urology research. Urothelial cells are primary transducers of physical and chemical stimuli. The urothelium has been shown to release various transmitters, such as ATP and ACh, in response to mechanical and chemical stimuli. These transmitters may regulate the activity of primary bladder afferent nerves, which are anatomically located in close proximity to the urothelium. In addition, lamina propria contains a dense layer of spindle-shaped cells categorized as myofibroblast or interstitial cells (ICs) and characterized by comparison with ICs of Cajal in the gastrointestinal tract. The role of bladder ICs has not yet been established, but they may constitute a structural and functional link between urothelial cells and sensory nerves and/or between urothelial cells and detrusor smooth muscle cells (Fig. 9.3).

In the urothelium-based hypothesis, changes in urothelial receptor function and neurotransmitter release, as well as changes in the sensitivity and coupling of the suburothelial myofibroblasts/interstitial cells, are suggested to contribute to increased afferent activity. This increased afferent activity amplifies the sensations of bladder fullness, thereby leading to urgency and predisposing to activation of the micturition reflex.

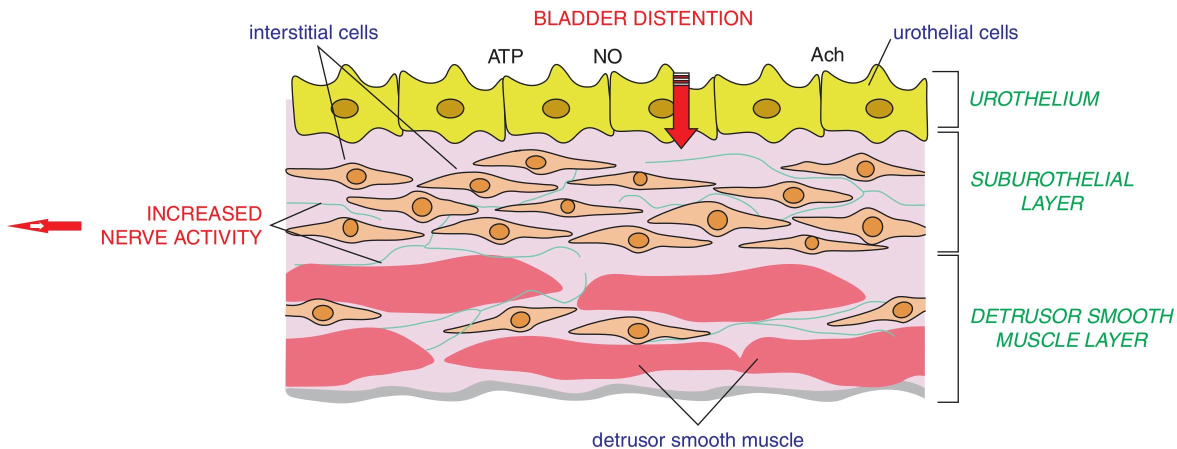


FIGURE 9.3 Urothelial signaling. A schematic view of the cellular structures in the different bladder layers. At present, there is still a debate on what precisely defines an interstitial cell in the human bladder. However, the stretch of the bladder during filling may evoke activity in afferent nerves directly or via interstitial cells

9.2.3 Abnormal Handling of Afferent Signals

As fully described in Chap. 1, in the past few decades, various methods of functional brain imaging have been used to study cerebral control of the bladder and urethra. Bladder control depends on an extensive network of brain regions. Dysfunction in various parts may contribute to Urgency incontinence suggesting that there are different phenotypes requiring different treatments.

Studies of older females with urgency incontinence, without DO, have linked bladder filling sensations such as desire to void and urgency to an exaggerated activation of the insula, the dorsal anterior cingulate cortex (ACC) and supplementary motor area (SMA). Similar activation is seen in healthy individuals, but with little awareness of bladder filling until a substantial volume of urine has accumulated, a desire to void is felt, and an opportunity to empty the bladder is sought. Conversely, the neural signature of DO itself seems to be a prefrontal cortex deactivation.

Clearly, different causes or combinations of causes may be responsible for Urgency incontinence in different individuals, implying that there are different phenotypes that may require different treatments. Understanding these differences and their causes will be the next great advance in diagnosis and therapy of this difficult problem.

9.3 Urodynamics in OAB-S

Urodynamic evaluation is potentially part of specialist assessment of OAB-S. It applies when conservative and medical management is suboptimal and when overactive bladder syndrome has a significant impact on a patient's quality of life. A major challenge in profiling patients with subtypes of idiopathic OAB is the lack of validated phenotypic markers to identify patients with different underlying pathophysiologies.

Note: Less than 20% of treatment trials profile patients according to suspected underlying pathophysiology of OAB. Detrusor overactivity on urodynamic testing is by far the most common categorization. The cost of subclassifying OAB by pathophysiologic condition would be far more expensive than a more "empiric" approach, and probably the treatment effects of current OAB therapies target a common symptomatic mechanism of urgency (i.e., DO) for different pathophysiologic conditions.

Urodynamically, patients with OAB-S can be stratified into clinical groups based on the presence or absence of involuntary detrusor contractions, the ability to abort contractions, and the ability to contract the urinary sphincter in response to contractions. Then, the urodynamic diagnoses associated with OAB symptoms should include at least three findings:

- Detrusor overactivity
- Increased filling sensation
- The ability or not to contract the external sphincter in response to detrusor behavior

Detrusor overactivity can be:

- *Phasic*, characterized by increasing amplitude in contractions when the bladder volume increases (Fig. 9.4). Detrusor overactivity is observed only in half of the patients with OAB symptoms**.** The association between OAB and DO appears to be more common in patients with urgency incontinence than in those without incontinence.

Note: If detrusor overactivity isn't observed during bladder filling, then a provocation test can be initiated, such as asking patient to cough, changing the position from sitting to standing, and/or generating the sound of running water.

- *Terminal*, a single involuntary detrusor contraction at cystometric capacity, causing incontinence often leading to bladder emptying (Fig. 9.5).

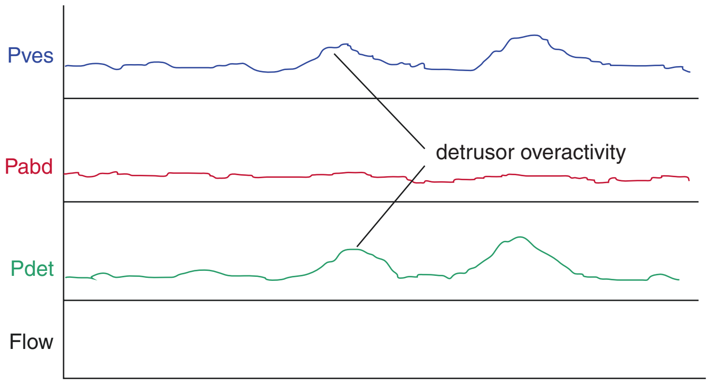


FIGURE 9.4 Phasic detrusor overactivity. Phasic DO is an involuntary detrusor contraction recognizable as a temporary increase in Pvess and Pdet. The ancient concept of minimum size of 5 cm H_2O has been updated. There is no minimum size for DO, but it should be just an observable increase of pressure

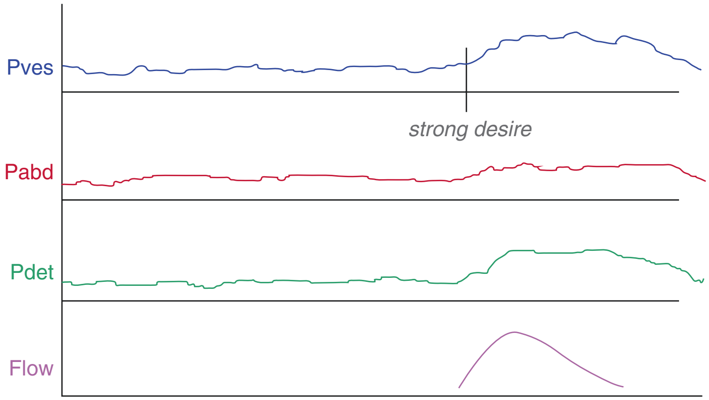


FIGURE 9.5 Terminal detrusor overactivity. Terminal DO is defined as a single involuntary contraction occurring at cystometric capacity which cannot be suppressed and usually results in a complete bladder emptying

The reduction of "warning time," i.e., the time between the compelling desire to void and the effective voiding, has been interpreted as the result of poor brain handling of afferent signals from the bladder. Timed voiding is probably the best therapeutic option for this group of patients.

In the absence of detrusor overactivity, the patient may report an *increased bladder sensation* during filling, that is, an early and persistent desire to void usually at a volume lower than 100 mL (Fig. 9.6). By some authors this finding is considered as the first step of a pain bladder syndrome (PBS). While urgency is the defining symptom of OAB and pain on bladder filling is the hallmark symptom of PBS, the relationship between urgency and pain, as well as the different afferent mechanisms associated with the genesis of these sensory symptoms, remains unknown. From a practical point of view, the efficacy of antimuscarinics is significantly lower in this group of patients.

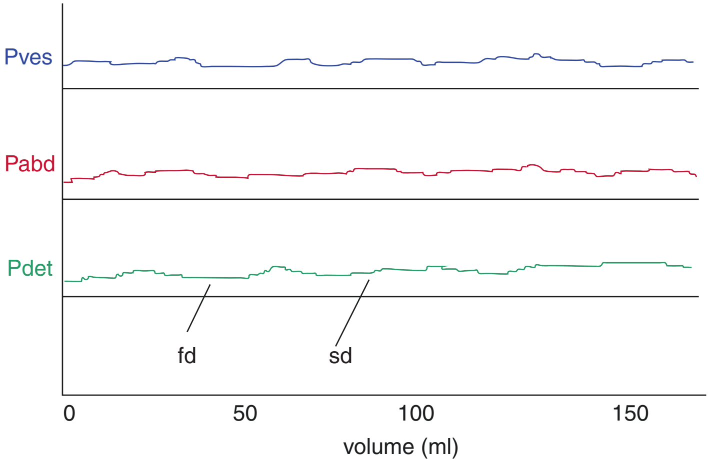


FIGURE 9.6 Increased bladder sensation. Bladder hypersensitivity is defined as an early first sensation of bladder filling which occurs at low bladder volume (usually lower than 100 mL) without any involuntary contraction

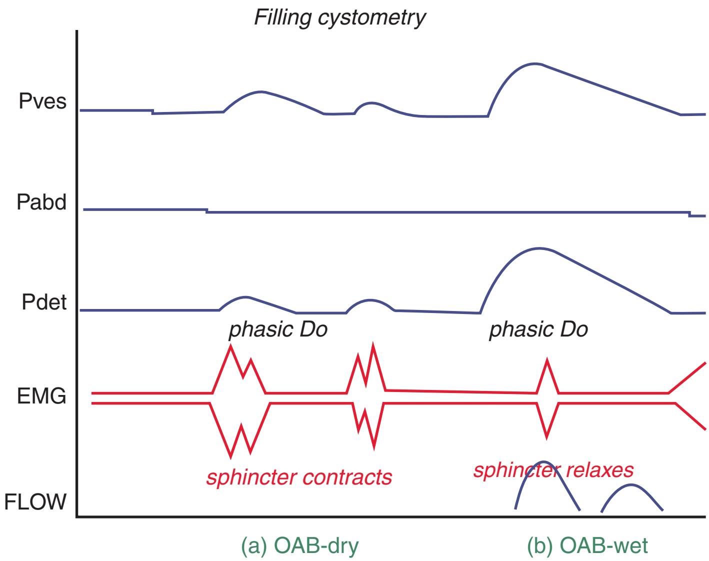


FIGURE 9.7 Sphincter behavior in OAB dry (**a**) and OAB wet (**b**). In OAB dry patient is aware of detrusor contraction and can voluntarily contract the sphincter aborting detrusor contraction and preventing incontinence. In OAB wet the patient is neither able to voluntarily contract the sphincter nor abort the detrusor contraction and voids involuntarily

Sphincter behavior during filling may account for an "OAB dry" (when patient is able to compensate detrusor contraction) or an "OAB wet" (when patient is unable to contract the sphincter) (Fig. 9.7).The inability to contract the sphincter may justify the practice of Kegel exercises.

In summary, it is likely that the OAB syndrome represents a spectrum of underlying pathophysiologic conditions. Unfortunately, few randomized clinical trials currently make an attempt to profile idiopathic OAB patients according to specific findings. In order to better target therapy based on pathophysiology, future studies should explore and validate standardized criteria for classification of idiopathic OAB subtypes.

9.4 OAB-S in Female: Key Notes for Clinical Assessment

- OAB is a syndrome (i.e., a symptom complex common to several clinical conditions).
- Comorbid conditions should be completely excluded.
- A urinalysis to rule out UTI and hematuria should be performed in any patient.
- In postmenopausal women, pelvic examination should exclude atrophic vaginitis and pelvic organ prolapse.
- Bladder diary may be useful in patients who cannot describe their voiding behavior.
- Post-void residual may be useful to exclude a voiding dysfunction and before antimuscarinics.
- Urodynamic testing is not indicated in the initial diagnostic workup.
- Urodynamic testing should be recommended in refractory patient who have failed multiple OAB treatments in order to identify the exact mechanisms of urgency. In particular, urodynamic testing should be recommended when invasive, potentially morbid and irreversible treatments are considered.

9.5 OAB-S in Female: Key Notes for Treatment (Fig. 9.8)

A detailed discussion of OAB treatments is beyond the scope of the chapter. Below are the most significant points:

- Initial management is conservative and includes education, bladder training, and advice on fluid intake. Drug therapy options include antimuscarinic medications (with ER formulations more efficacious than the correspondent IR formulations) and beta-3 adrenergic receptor agonists.
- Persistent symptoms may require adjustment of medication including association of different drugs.

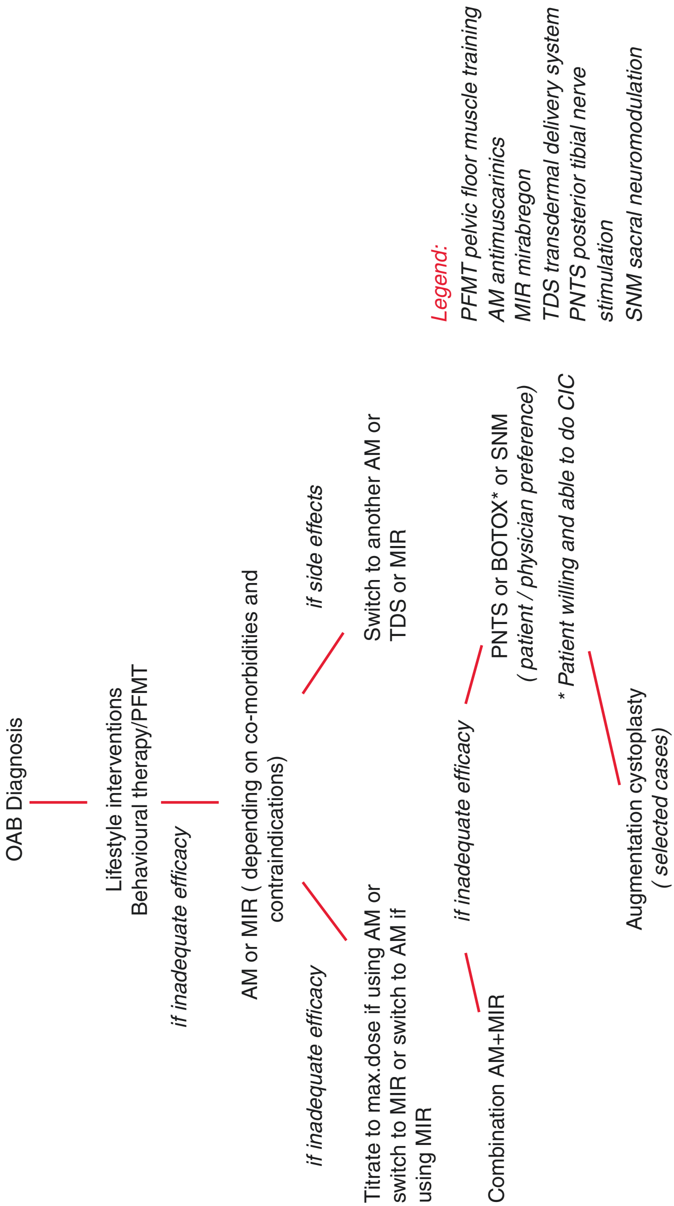


FIGURE 9.8 OAB treatment algorithm

- There is considerable variation in the current guidelines regarding the use of oral pharmacotherapy, with antimuscarinics. The discrepancies reflect the lack of adequate comparative head-to-head studies between AMs as well as of well-designed pharmacoeconomic studies. The EAU Guidelines 2015 propose the use of either immediate-release (IR) or extended-release (ER) formulations as first-line drugs, while AUA/SUFU Guidelines favor the ER formulations over the IR ones and NICE Guidelines (largely cost-driven) favor mostly IR formulations. Likewise, there is no unanimity concerning the use of the new beta-3 agonist mirabegron, and no recommendations currently stand on combination pharmacotherapy.

For refractory cases (ineffective treatment with AMs for 3 months or intolerance due to side effects), BoNT/A bladder injections can be used, provided the patient is able and willing to do intermittent self-catheterization, which is necessary in about 5% of treated patients.

- Bladder instillation of liposome-encapsulated BoNT/A is a new approach, deserving further research.
- Posterior tibial nerve stimulation and sacral nerve stimulation are other approaches. *(See* Chap. 12 *for further details.)*
- Major reconstructive surgery, such as auto-augmentation via detrusor myectomy or augmentation cystoplasty with the use of bowel, is rarely undertaken in modern practice but remains a possibility in extreme cases.

Suggested Readings

Overviews

Truzzi JC, Gomes CM, Bezerra CA, Plata IM, Campos J, Garrido GL, et al. Overactive bladder—18 years—Part I. Int Braz J Urol. 2016a;42:188–98.

Truzzi JC, Gomes CM, Bezerra CA, Plata IM, Campos J, Garrido GL, et al. Overactive bladder—18 years—Part II. Int Braz J Urol. 2016b;42:199–214.

Wallace KM, Drake MJ. Overactive bladder. F1000Res. 2015;4. pii: F1000 Faculty Rev-1406.

Gormley EA, Lightner DJ, Faraday M, Vasavada SP, American Urological Association.; Society of Urodynamics, Female Pelvic Medicine. Diagnosis and treatment of overactive bladder (non-neurogenic) in adults: AUA/SUFU guideline amendment. J Urol. 2015;193:1572–80.

Nambiar A, Lucas M. Chapter 4: guidelines for the diagnosis and treatment of overactive bladder (OAB) and neurogenic detrusor overactivity (NDO). Neurourol Urodyn. 2014;33(Suppl 3):S21–5.

Haab F. Chapter 1: the conditions of neurogenic detrusor overactivity and overactive bladder. Neurourol Urodyn. 2014;33(Suppl 3):S2–5.

Robinson D, Cardozo L. Overactive bladder: diagnosis and management. Maturitas. 2012;71:188–93.

Pathophysiology

Chapple C. Chapter 2: pathophysiology of neurogenic detrusor overactivity and the symptom complex of "overactive bladder". Neurourol Urodyn. 2014;33(Suppl 3):S6–13.

Gajewski JB, Kanai AJ, Cardozo L, Ykeda Y, Zabbarova IV. Does our limited knowledge of the mechanisms of neural stimulation limit its benefits for patients with overactive bladder? ICI-RS 2013. Neurourol Urodyn. 2014;33:618–21.

Hanna-Mitchell AT, Kashyap M, Chan WV, Andersson KE, Tannenbaum C. Pathophysiology of idiopathic overactive bladder and the success of treatment: a systematic review from ICI-RS 2013. Neurourol Urodyn. 2014;33:611–7.

Chacko S, Cortes E, Drake MJ, Fry CH. Does altered myogenic activity contribute to OAB symptoms from detrusor overactivity? ICI-RS 2013. Neurourol Urodyn. 2014;33:577–80.

Kitta T, Mitsui T, Kanno Y, Chiba H, Moriya K, Shinohara N. Brain-bladder control network: the unsolved 21st century urological mystery. Int J Urol. 2015;22:342–8.

Morris CL. Urge urinary incontinence and the brain factor. Neurourol Urodyn. 2013;32:441–8.

Tadic SD, Griffiths D, Schaefer W, Murrin A, Clarkson B, Resnick NM. Brain activity underlying impaired continence control in older women with overactive bladder. Neurourol Urodyn. 2012;31:652–8.

Chai TC, Russo A, Yu S, Lu M. Mucosal signaling in the bladder. Auton Neurosci. 2015;200:1566–10702.

Andersson KE, McCloskey KD. Lamina propria: the functional center of the bladder? Neurourol Urodyn. 2014;33:9–16.

Kanai A, Fry C, Hanna-Mitchell A, Birder L, Zabbarova I, Bijos D, Ikeda Y. Do we understand any more about bladder interstitial cells? -ICI-RS 2013. Neurourol Urodyn. 2014;33:573–6.

Birder LA, Andersson KE, Kanai AJ, Hanna-Mitchell AT, Fry CH. Urothelial mucosal signaling and the overactive bladder-ICI-RS 2013. Neurourol Urodyn. 2014;33:597–601.

Castro-Diaz D, Cardozo L, Chapple C. Urgency and pain in patients with overactive bladder and bladder pain syndrome. What are the differences? Int J Clin Practice. 2014;68:356–62.

De Wachter SG, Heeringa R, van Koeveringe GA, et al. On the nature of bladder sensation: the concept of sensory modulation. Neurourol Urodyn. 2011;30:1220–6.

Banakhar MA, Al-Shaiji TF, Hassouna MM. Pathophysiology of overactive bladder. Int Urogynecol J. 2012;23:975–82.

Roosen A, Chapple CR, Dmochowski RR, et al. A refocus on the bladder as the originator of storage lower urinary tract symptoms: a systematic review of the latest literature. Eur Urol. 2009;56:810–9.

Diagnosis

Bhide AA, Cartwright R, Khullar V, Digesu GA. Biomarkers in overactive bladder. Int Urogynecol J. 2013;24:1065–72.

Scarpero H. Urodynamics in the evaluation of female LUTS when are they helpful and how do we use them? Urol Clin North Am. 2014;41:429–38.

Rovner ES, Goudelocke CM. Urodynamics in the evaluation of overactive bladder. Curr Urol Rep. 2010;11:343–7.

Rutman MP, Cha DY, Blaivas JG. How do urodynamics findings influence the treatment of the typical patient with overactive bladder? Curr Urol Rep. 2012;13(5):370–8.

Rosier PF, Giarenis I, Valentini FA, Wein A, Cardozo L. Do patients with symptoms and signs of lower urinary tract dysfunction need a urodynamic diagnosis? ICI-RS 2013. Neurourol Urodyn. 2014;33:581–6.

Flisser AJ, Blaivas JG. Role of cystometry in evaluating patients with overactive bladder. Urology. 2002;60(Suppl 1):33–42.

Al-Zahrani AA, Gajewski J. Urodynamic findings in women with refractory overactive bladder symptoms. Int J Urol. 2016;23:75–9.

Treatment

Apostolidis A, Averbeck MA, Sahai A, Rahnamai's MS, Anding R, Robinson D, et al. Can we create a valid treatment algorithm for patients with drug resistant overactive bladder (OAB) syndrome or detrusor overactivity (DO)? Results from a think tank (ICI-RS 2015). Neurourol Urodyn. 2017;36:882–93.

Vouri SM, Kebodeaux CD, Stranges PM, Teshome BF. Adverse events and treatment discontinuations of antimuscarinics for the treatment of overactive bladder in older adults: a systematic review and meta-analysis. Arch Gerontol Geriatr. 2017;69:77–96.

Olivera CK, Meriwether K, El-Nashar S, Grimes CL, Chenn CC, Orejuela F, et al. Nonantimuscarinic treatment for overactive bladder: a systematic review. Am J Obstet Gynecol. 2016;215:34–57.

Visco AG, Fraser MO, Newgreen D, Oelke M, Cardozo L. What is the role of combination drug therapy in the treatment of overactive bladder? ICI-RS 2014. Neurourol Urodyn. 2016;35:288–92.

Madhuvrata P, Singh M, Hasafa Z, Abdel-Fattah M. Anticholinergic drugs for adult neurogenic detrusor overactivity: a systematic review and meta-analysis. Eur Urol. 2012;62:816–30.

Linsenmeyer TA. Use of botulinum toxin in individuals with neurogenic detrusor overactivity: state of the art review. J Spinal Cord Med. 2013;36:402–19.

Andersson KE. New pharmacologic targets for the treatment of the overactive bladder: an update. Urology. 2004;63:32–41.

Chapter 10
UDS in Voiding Dysfunction Syndrome (VD-S)

10.1 Background

Although emptying problems are more commonly reported in men, a significant number of women also complain of voiding dysfunction (VD). Despite the recent advances in the standardization of terminology of lower urinary tract dysfunction at present, there is a lack of consensus regarding a precise diagnosis and definition of voiding abnormalities in women.

According to the latest terminology report from ICS, voiding dysfunction (VD) is defined as an "abnormally slow and/or incomplete micturition, based on symptoms and urodynamic investigations."

Note: As mentioned before (Chap. 1), the voiding mechanism in female is increasingly recognized as more complex than in men. The conventional idea of a detrusor contraction has been overtaken by various studies showing women void via a variety of mechanisms, including abdominal straining plus pelvic floor relaxation, with or without a detrusor contraction.

Emptying problems in women encompass a complex of complaints characterized by poor and/or intermittent stream, sensations of incomplete emptying, double voiding, and possibly hesitancy and terminal dribbling. These symptoms rarely

G. Vignoli, *Urodynamics for Urogynecologists*,
https://doi.org/10.1007/978-3-319-74005-8_10

exist in isolation and often are found in association with other storage-related symptoms such as frequency, urgency, and nocturia. In addition recurrent UTIs and urinary incontinence may be present.

10.2 Prevalence of Emptying Problems in Women

The evidence from two epidemiological studies (EPIC, EpiLUTS) suggest that, overall, voiding problems are less common in women than in men.

The prevalence varies from 5 to 10% depending on the definition of post-void residual used. It would seem reasonable to consider a post-void residual of >100 ml to be significant although many women may still remain asymptomatic with a PVR of >200 ml, and hence it is imperative to consider the clinical context.

10.3 Causes of Voiding Dysfunction in Females

The causes of female VD can be broadly divided into detrusor underactivity (DU) and bladder outflow obstruction (BOO) which may either be anatomical and functional (Box 10.1).

Classically, diminished bladder emptying has been reported to occur because of decreased quality of detrusor contractility or impairment of the outflow tract. In some cases a combination of both may coexist.

Recent studies, however, have demonstrated that impaired voiding is more than a simply issue of contractile properties of detrusor smooth muscle or outflow obstruction but is a complex interplay of sensory function and central processing of afferent signals by the brain, which implement the detrusor muscular function and the resistance offered by the lower urinary tract (Fig. 10.1).

Box 10.1: Causes of Female Voiding Dysfunction

- Detrusor underactivity
  - Myogenic (age)
  - Neurogenic (diabetes)
  - Iatrogenic (radical pelvic surgery)
  - Idiopathic
- Bladder outlet obstruction

Anatomical

  - Anti-incontinence surgery
  - POP

Functional

- – Dysfunctional voiding (including Fowler's syndrome)
- Mixed forms

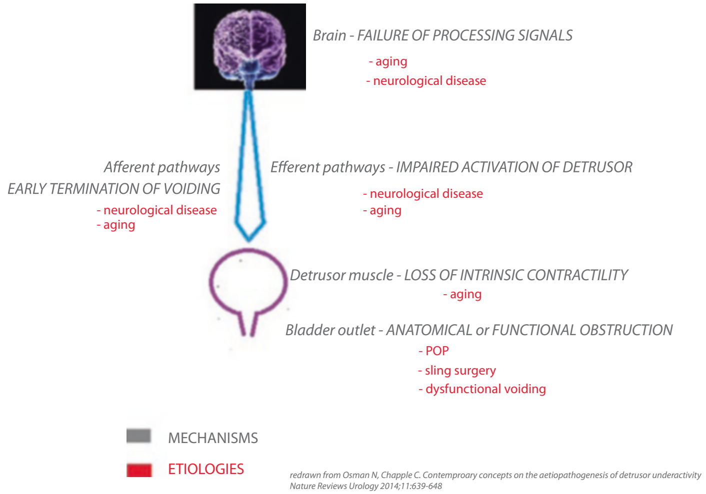


FIGURE 10.1 Major etiologies sites of dysfunction and pathogenic mechanisms that result in DU

Understanding the pathophysiology of voiding dysfunction is a necessary step to interpret the diagnostic procedures and provide appropriate treatment.

10.4 Detrusor Underactivity

The International Continence Society defines *detrusor underactivity* (DU) as a "contraction of reduced strength and/or duration, resulting in prolonged bladder emptying and/or a failure to achieve complete bladder emptying within a normal time span" and *underactive bladder* (UAB) as a symptom complex suggestive of detrusor underactivity, usually characterized by prolonged micturition time with or without a sensation of incomplete bladder emptying, usually with hesitancy, reduced sensation on filling, and a slow stream.

DU is observed in patients with pathological conditions such as diabetic neuropathy or bladder dysfunction after surgeries of intra-pelvic organs. However, DU frequently occurs without obvious etiology, suggesting that, in some cases, it may be a distinct disease entity. Detrusor overactivity sometimes coexists with DU in the elderly population. The condition is called detrusor hyperactivity with impaired contractility (DHIC).

10.4.1 Detrusor Underactivity and Aging

Bladder contractility is reduced by 35% in women between 50 and 80 years of age. Uroflow studies have demonstrated an age-dependent decrease in Qmax which is similar in both sexes. Increasing age is associated with an increase of the collagen: muscle ratio in bladder wall that is reflected in a decline of contractility. Specific ultrastructural features described as "degeneration patterns" have been described in the bladder of elderly people including shrinked appearance of the cells, disrupted sarcolemma, and deposition of collagen in the interstitium.

Note: As well as in elderly, “degeneration patterns” have been described in diabetic patients and in patients who undergone extended pelvic surgery like radical hysterectomy, or anterior/abdominoperineal resection, and thus may be considered neurogenic in origin.

Recently, attention has been focused on bladder ischemia as a common pathophysiologic mechanism for age-related bladder dysfunction, including DO and DU. Atherosclerosis is one of the important causes of reduction of blood flow of the bladder, leading to chronic bladder ischemia. Studies in animal models suggest that the extent of bladder dysfunction in chronic bladder ischemia depends on the degree and duration of ischemia. Moderate ischemia may cause DO and OAB symptoms via sensitization of afferent pathways. When bladder ischemia becomes severe, progression of damage to nerves (both afferent and efferent) and detrusor muscle may cause DO and inability to empty the bladder.

Finally, an abnormal handling of afferent bladder signals by the brain, frequently reported in association with increasing age, may contribute to DU. To void efficiently, a feed-forward mechanism by which urinary flow in the urethra helps to enhance and maintain adequate contractile function of the bladder, until the bladder is empty is required. FMRI in older people with voiding dysfunction found diminished response to bladder filling in the insula, the area of the brain responsible for mapping visceral sensations.

10.5 Obstruction

Bladder outlet obstruction (BOO) is defined by the ICS as a “generic term for obstruction during voiding.” Although BOO is one of the most common clinical complaints in adult men, it is a condition less common in women. BOO in women is subdivided by cause into anatomic and functional subgroups.

Anatomic causes are generally more obvious than functional causes and include:

- Previous anti-incontinence surgery (MUS)
- Pelvic organ prolapse

10.5.1 Obstruction and Previous MUS Surgery

One potential complication of mid-urethral sling surgery is postoperative voiding dysfunction, with reported rates varying between 5 and 20% and need of surgical re-intervention in 0–4% of patients. Because symptoms are misleading and not indicative of urinary obstruction, diagnosis and subsequent treatment are often late. Peculiar symptoms of obstruction are the need to immediately re-void and position-dependent micturition. However they are often masked by OAB symptoms or recurrent UTI. Pressure-flow study is the only way to diagnose an obstruction (see below).

Surgery, generally performed through vaginal route, includes simple transection or partial removal of the sling at the surgeon's discretion at the time of surgery. However, there is still an active discussion on how to optimally treat this condition. Satisfaction rate is around 75%. Dissatisfied patient complains both recurrent SUI and OAB symptoms. Performing surgical release more than 180 days after obstructive anti-incontinence surgery seems associated with less recurrent SUI, while a surgical release before 70 days seems to have a decrease of postoperative OAB symptoms.

10.5.2 Obstruction and POP

Prolapse greater than Stage 2 is sufficient to cause a lack of proximal urethral funnelling with urethral kinking that can result in bladder outlet obstruction. Again the diagnosis of obstruction is done during a pressure-flow study (see below).

Note: In some cases of POP, urethral kinking may be difficult to measure or observe even under fluoroscopy because

the catheter tends to straighten the urethra during the pressure/flow investigation.

There is an overall agreement that prolapse-induced bladder outlet obstruction may trigger bladder OAB symptoms. Surgical management of prolapse leads to improvements of OAB symptoms in most of the patients. The topic is widely discussed in Chap. 8.

Functional—Functional obstruction is caused by abnormal contraction of the periurethral muscles or failure to relax them during voiding. Functional obstruction includes:

- Dysfunctional voiding
- Fowler's syndrome

10.5.3 Dysfunctional Voiding (DV)

DV is defined as "an intermittent and/or fluctuating flow rate due to involuntary intermittent contractions of the periurethral striated muscle during voiding in neurologically normal individuals". In contrast to detrusor sphincter dyssynergia, there is no neurologic cause for the findings. DV appears to be a learned behavior since in physiologic voiding, the pelvic floor muscles and external striated sphincter should relax just prior to detrusor contraction. A precise urodynamically quantifiable definition does not exist for DV, and urethral voiding dysfunction is usually diagnosed on the basis of abnormalities of flow and pelvic floor EMG, with either Qmax reduced or a flow curve interrupted or fluctuating due to the persistence of EMG activity during voiding (Fig. 10.2).

Note: Difficulty in initiating the voiding reflex may occur as a consequence of embarrassment or mental stress in the urodynamic room, the so-called "bashful" or "shy bladder" syndrome. In these conditions flow rate may not be representative for the actual physiology of the lower urinary tract of the women. Not much is known about the prevalence of psychogenic voiding dysfunction and its influence on the final diagnosis among patients referred to urodynamic clinic.

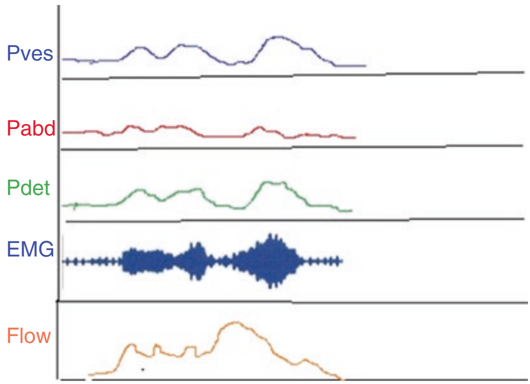


FIGURE 10.2 Urodynamic findings in DV. During voiding there is a patchy increase of EMG activity that induces a fluctuating stream

Dysfunctional voiding is essentially a problem of striated musculature. The relative importance of urethral smooth muscle in women with voiding dysfunction is unclear. There is no histological evidence of sympathetic alpha-adrenergic receptors in the female urethra nor for its significance at the bladder neck level. Alpha-blockers have never consistently demonstrated objective effect on female voiding dysfunction despite some reports on subjective improvement of micturition

10.5.4 Fowler's Syndrome

Another type of urethral abnormality that can result in functional BOO is Fowler's syndrome. Fowler's syndrome typically presents in young women as painless urinary retention (often in excess of 1 L) with no neurologic or anatomic etiology. It is not known if Fowler's syndrome represents a subgroup of patients with VD or a distinct clinical entity in young women with chronic urinary retention. The diagnosis is made by a characteristic electromyography pattern that demonstrates failure of urethral sphincter relaxation and decrease in bladder sensation.

10.6 Assessment of Voiding Dysfunction in Female

"The female bladder is an even less reliable witness."

Most of the patients complain both storage and voiding symptoms. The predictive value of voiding symptoms has been shown to be poor when predicting female VD. Women are most concerned with leakage than with retention and generally fail to describe their stream and compare it with the past. Recurrent UTI may be a red flag symptom.

The diagnosis of emptying problems in women relies mostly on urodynamics although the extent of investigation is dependent upon the clinical setting. All women complaining of difficulty in micturition should be investigated first with a free flowmetry and post-void residual. If abnormal, filling cystometry with a pressure-flow study will help to determine whether the cause is detrusor underactivity or outlet obstruction.

10.7 Urodynamic Challenges in the Diagnosis of Female DU/BOO

To date, there are no universally accepted criteria to diagnose and quantify obstruction and detrusor underactivity in women.

10.7.1 Obstruction

Several definitions for female BOO have been proposed using various combinations of clinical parameters, pressure-flow criteria, and radiographic evidence of BOO (see Box 10.2).

There is a general agreement that absolute pressure and flow values are imprecise, and urodynamic tracings alone are insufficient to diagnose BOO in women. Probably only

Box 10.2: Urodynamics Criteria for Diagnosing BOO in Women

Author	Qmax	Pdet@Qmax
Axelrod and Blaivas	<12 mL/s	> 20 cmH_2O
Massey and Abrams	<15 mL/s	> 20 cmH_2O
Chassagne	<15 mL/s	> 20 cmH_2O
Lemack and Zimmern (2000)	< 11 mL/s	> 21 cmH_2O
Lemack and Zimmern (2004)	<12 mL/s	> 25 cmH_2O
Nitti	*fluoroscopic evidence of obstruction with any low flow and high Pdetmax*	

fluoroscopic evidence of obstruction between the bladder neck and distal urethra in the presence of a sustained detrusor contraction of any magnitude, usually associated with a reduced or delayed flow, has the highest concordance with a clinical suspicion of obstruction.

Only recently has been reported (ICI-RS 2014) that the criterion *Pdetmax > 2Qmax* is the best determinant of BOO in women.

Note: Technically, a proportion of women may be unable to void during urodynamic testing, and concerns have been expressed that the use of certain urethral catheter sizes during pressure-flow study may lead to over diagnosis by increasing outlet resistance.

10.7.2 Detrusor Underactivity

Bladder contractility in women has received less attention than in male.

The isovolumetric pressure attained when flow is interrupted (the stop test) provides in theory a good estimate of

contraction strength (Coolsaet). However, mechanical interruption of flow by occlusion with a balloon catheter may be difficult to perform and may induce discomfort in the patient. Therefore others methods of estimating bladder contractility have been proposed.

Several algorithms have been proposed to quantify detrusor power during voiding, like the Griffiths' Watt factor, Schafer's nomogram, and bladder contractility index.

All are based on the bladder output relation (BOR), an inverse relation between pressure and flow that is analogous to Hill's equation for contracting muscle. If the slope and curvature of the BOR are known, the isovolumetric detrusor pressure can be estimated by extrapolating (projecting) the actual pressure back to the pressure axis. PIP (projected isovolumetric pressure) can be calculated by the formula PIP = Pdet + KQ where Q is the maximum flow rate and Pdet is the corresponding pressure (Fig. 10.3).

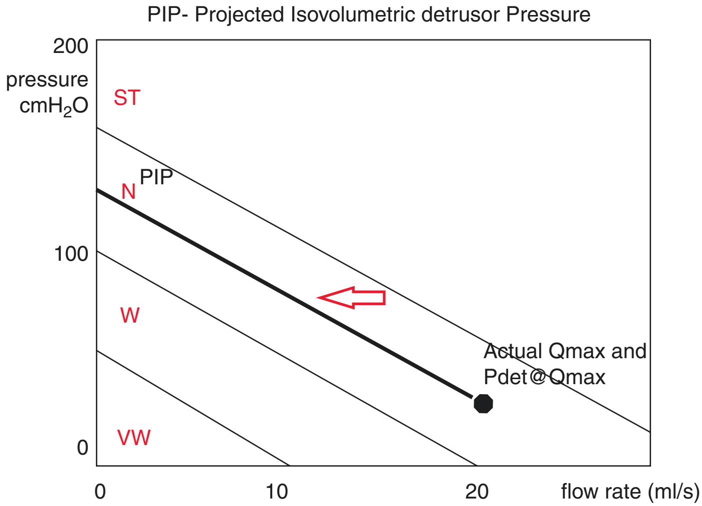


Figure 10.3 Projected isovolumetric pressure. The isovolumetric detrusor pressure can be estimated by extrapolating (projecting) the actual pressure back to the pressure axis

Schafer took K to be 5 cmH_2O/mL/s and suggested that values of PIP greater than150 cmH_2O represented strong contractions (ST); values from 100 to 150 cmH_2O, normal contractions (N); values from 50 to 100 cmH_2O, weak contractions (W); and values below 50 cmH_2O, very weak contractions (VW). He constructed his well-known contractility nomogram that together with possible information on bladder outlet obstruction allows contraction strength to be classified in one of these classes (Fig. 10.4).

The problems with these methods are that they are calculated for male pressure-flow study and when applied in female tend to overestimate the value of bladder contractility. The female void with lower detrusor pressure and higher flow rate. Adjustments of PIP have been proposed, modifying the value of constant K in calculation of female bladder contractility from $K = 5$ to $K = 1$ cmH_2O/mL/s, thus obtaining more reliable estimates of bladder contractility in females still maintaining the categories proposed for men.

To date, there is only one validated nomogram for women, the Blavais-Groutz nomogram, which, however, does not

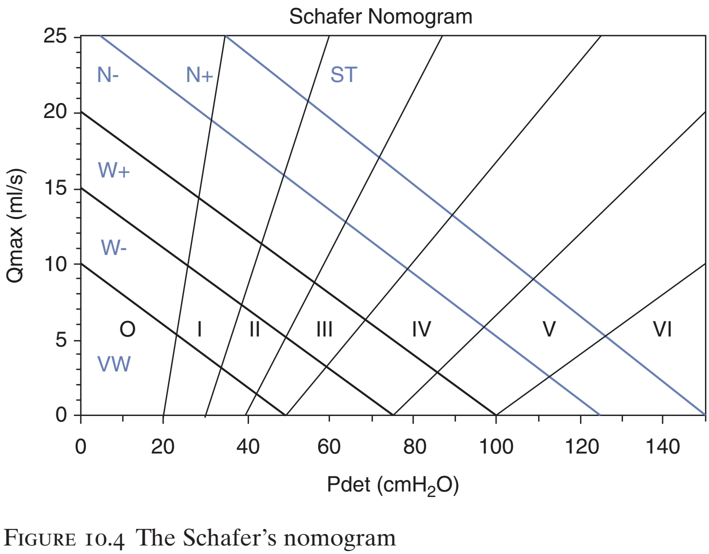


FIGURE 10.4 The Schafer's nomogram

consider detrusor contractility. The nomogram identifies three degrees of obstruction but has received criticism concerning its sensitivity, particularly in the mild obstruction zone (Fig. 5.4). Blaivas and Groutz described as important the obstructive effect of 7Fr urethral catheters accounting for misleading results of pressure/flow curves in women. They proposed using the value of maximum flow from the non-intubated free flowmetry just previous to cystometry plotted against the maximum detrusor pressure (Pdetmax) instead of detrusor pressure at the maximum flow (Pdet@Qmax) since they found no statistical differences between Pdetmax and Pdet@Qmax. In addition Pdetmax can be used even in absence of flow, fairly common in women (see note).

Note: As additional problem, the vast majority of computer-urodynamic investigations are performed as in-office procedures; however, there is ongoing debate about their universal validity because relevant urethral sphincter activity may interfere with voiding. Recently it has been reported that 84% of patients with acontractile detrusor during office urodynamics have detrusor contractions during ambulatory urodynamics. Therefore, in patients with VD-S, results of conventional in-office urodynamics should be compared, when possible, with ambulatory urodynamics to avoid misinterpretations.

10.8 Keynotes on Clinical Diagnosis of VD in Women

1. Mixed symptoms (storage and voiding)
2. Recurrent UTI
3. Interrupted flow
4. PVR > 100 ml
5. Qmax <12–15 mL/s
6. Pdet@Qmax >20–25 cmH_2O
7. Pdetmax>2Qmax
8. Videourodynamics: mid-urethral narrowing during voiding
9. Dysfunctional voiding: increase in EMG activity during voiding

10.9 Keynotes on Clinical Management of VD in Women

The management of women with voiding dysfunction should be individualized to each patient.

The following clinical scenarios can be outlined:

1. Patient asymptomatic with low PVR: double voiding and periodic PVR measurements is all that is required.
2. Patient with mixed symptoms and low PVR: antimuscarinic therapy with frequent residual assessments to ensure voiding function is not deteriorating.
3. Patient with voiding dysfunction following anti-incontinence surgery:
   - Short-term urethral catheter since evidence suggest that <5% of patients require catheterization for more than 1 week.
   - Women with persistent symptoms: short-term CIC since the majority of postoperative voiding difficulties will resolve, and by delaying intervention the risk of recurrent stress incontinence (SUI) would appear to be reduced.
   - Women with persistent symptoms, who are unwilling to self-catheterize: urethral dilatation or urethrolysis. Both procedures improve symptoms (at least in the short period), but SUI may recur.
4. Patient with voiding dysfunction and POP: surgical correction.

   Evidence suggest that surgical correction of urogenital prolapse is effective both in improving symptoms of VD and those concomitant with storage phase.
5. Dysfunctional voiding: pelvic floor rehabilitation.

   In a small Italian study, treatment with tamsulosin resulted in a significant improvement in symptoms (62.5% of pts) implying that pharmacological therapy may have a role in management of dysfunctional voiding.

Suggested Readings

Overviews

Hoffman DS, Nitti VW. Female Bladder Outlet Obstruction. Curr Urol Rep. 2016;17:31.

King AB, Goldman HB. Bladder outlet obstruction in women: functional causes. Curr Urol Rep. 2014;15:436.

Robinson D, Staskin D, Laterza RM, Koelbl H. Defining female voiding dysfunction: ICI-RS 2011. Neurourol Urodyn. 2012;31:313–6.

Tunuguntla HS, Tunuguntla R, Barone J, Kanagarajah P, Gousse AE. Voiding dysfunction in the female patient: is the "syndrome" paradigm valid? Curr Urol Rep. 2011;12:377–80.

Abdel Raheem A, Madersbacher H. Voiding dysfunction in women: how to manage it correctly. Arab J Urol. 2013;11:319–30.

Cohn JA, Brown ET, Kaufman MR, Dmochowski RR, Reynolds WS. Underactive bladder in women: is there any evidence? Curr Opin Urol. 2016;26:309–14.

Robinson D, Staskin D, Laterza RM, Koelbl H. Defining female voiding dysfunction: ICI-RS 2011. Neurourol Urodyn. 2012;31:313–6.

Epidemiology

Irwin DE, Milson I, Hunskaar S, Reilly K, Kopp Z, Herschorn S, et al. Population based survey of urinary incontinence, overactive bladder, and other lower urinary tract symptoms in five countries: results of the EPIC study. Eur Urol. 2006;50:1306–14.

Coyne KS, Sexton CC, Thompson CL, Milsom I, Irwin D, Kopp ZS, et al. The prevalence of lower urinary tract symptoms (LUTS) in the USA, UK and Sweden: results from the epidemiology of LUTS (EpiLUTS) study. BJU Int. 2009;104:352–60.

Clinical Assessment

Gammie A, Kirschner-Hermanns R, Rademakers K. Evaluation of obstructed voiding in the female: how close are we to a definition? Curr Opin Urol. 2015;25:292–5.

Espuña-Pons M, Cardozo L, Chapple C, Sievert KD, van Kerrebroeck P, Kirby MG. Overactive bladder symptoms and voiding dysfunction in neurologically normal women. Neurourol Urodyn. 2012;31:422–8.

Lowenstein L, Anderson C, Kenton K, Dooley Y, Brubaker L. Obstructive voiding symptoms are not predictive of elevated post void residual urine volumes. Int Urogynaecol J Pelvic Floor Dysfunct. 2008;19:801–4.

Al-Sharani M, Lovatis D. Do subjective symptoms of obstructive voiding correlate with the post void residual urine in women? Int Urogynaecol J. 2008;16:12–4.

Gehrich A, Stany MP, Fischer JR, Buller J, Zahn CM. Establishing a mean postvoid residual volume in asymptomatic perimenopausal and postmenopausal women. Obstet Gynaecol. 2007;110:827–32.

Luckacz ES, DuHamel E, Menefee SA, Luber KM. Elevated post-void residual in women with pelvic floor disorder: prevalence and associated risk factors. Int Urgynaecol J. 2007;18:397–400.

Urodynamics

Rosier P, GaJewski J, Sand P, Szabó L, Capewell A, Hosker G. Executive Summary: The International Consultation on Incontinence 2008—Committee on: "Dynamic Testing"; for urinary incontinence and for fecal incontinence. Part 1: innovations in urodynamic techniques and urodynamic testing for signs and symptoms of urinary incontinence in female patients. Neurourol Urodyn. 2010;29:140–5.

Axelrod S, Blaivas J. Bladder neck obstruction in women. J Urol. 1987;137:497–9.

Massey JA, Abrams PH. Obstructed voiding in the female. Br J Urol. 1988;61:36–9.

Chassagne S, Bernier PA, Haab F, Roehrborn CG, Reisch JS, Zimmern PE. Proposed cutoff values to define bladder outlet obstruction in women. Urology. 1998;51(3):408–11.

Lemack GE, Zimmern PE. Pressure flow analysis may aid in identifying women with outflow obstruction. J Urol. 2000;163(6):1823–8.

Defreitas GA, Zimmern PE, Lemack GE, Shariat SF. Refining diagnosis of anatomic female bladder outlet obstruction: comparison of pressure-flow study parameters in clinically obstructed women with those of normal controls. Urology. 2004;64(4):675–9.

Nitti VW, Tu LM, Gitlin J. Diagnosing bladder outlet obstruction in women. J Urol. 1999;161:1535–40.

Rademakers K, Apostolidis A, Constantinou C, Fry C, Kirschner-Hermanns R, Oelke M, et al. Recommendations for future development of contractility and obstruction nomograms for women. ICI-RS 2014. Neurourol Urodyn. 2016;35:307–11.

Groutz A, Blaivis JG, Chaikin DC. Bladder outlet obstruction in women: definition and characteristics. Neurourol Urodyn. 2000;19:213–20.

Groutz A, Blaivas JG, Sassone AM. Detrusor pressure uroflowmetry studies in women: effect of a 7Fr transurethral catheter. J Urol. 2000;164:109–14.

Massolt ET, Groen J, Vierhout M. Application of the Blaivas–Groutz bladder outlet obstruction nomogram in women with urinary incontinence. Neurourol Urodyn. 2005;24:237–42.

Kuo HC. Videourodynamic characteristics and lower urinary tract symptoms of female bladder outlet obstruction. Urology. 2005;66:1005–9.

Detrusor Underactivity

Osman N, Mangera A, Hillary C. The underactive bladder: detection and diagnosis. F1000Res. 2016;25:5; pii: F1000 Faculty Rev-102.

Cohn JA, Brown ET, Kaufman MR, Dmochowski RR, Stuart Reynolds W. Underactive bladder in women: is there any evidence? Curr Opin Urol. 2016;26:309–14.

Smith P, Birde L, Abrams P, Wein A, Chapple C. Detrusor underactivity and the underactive bladder: symptoms, function, cause—what do we mean? ICI-RS think tank 2014. Neurourol Urodyn. 2016;35:312–7.

Park J, Lavelle JP, Palmer MH. Voiding dysfunction in older women with overactive bladder symptoms: a comparison of urodynamic parameters between women with normal and elevated post-void residual urine. Neurourol Urodyn. 2016;35:95–9.

Osman NI, Chapple CR, Abrams P, Dmochowski R, Haab F, Nitti V, et al. Detrusor underactivity and the underactive bladder: a new clinical entity? A review of current terminology, definitions, epidemiology, aetiology, and diagnosis. Eur Urol. 2014;65:389–98.

Yoshida M, Yamaguchi O. Detrusor underactivity: the current concept of the pathophysiology. Low Urin Tract Symptoms. 2014;6:131–7.

Osman NI, Chapple CR, Abrams P, Dmochowski R, Haab F, Nitti V, et al. Detrusor underactivity and the underactive bladder: a new clinical entity? A review of current terminology, definitions, epidemiology, aetiology, and diagnosis. Eur Urol. 2014;65:389–98.

Osman NI, Chapple CR. Contemporary concepts in the aetiopathogenesis of detrusor underactivity. Nat Rev. Urol. 2014;11:639–48.

Koeveringe G, Rademakers K, Birder L, Korstanje C, Daneshgari F, Ruggieri MR, et al. Detrusor underactivity: pathophysiological considerations, models and proposals for future research. ICI-RS 2013. Neurourol Urodyn. 2014;33:591–6.

van Koeveringe GA, Vahabi B, Andersson KE, Kirschner-Herrmans R, Oelke M. Detrusor underactivity: a plea for new approaches to a common bladder dysfunction. Neurourol Urodyn. 2011;30:723–8.

van Koeveringe GA, Rahnama'i MS, Berghmans BC. The additional value of ambulatory urodynamic measurements compared with conventional urodynamic measurements. BJU Int. 2010;105:508–13.

Griffiths D, Tadic SD, Schaefer W, Resnick NM. Cerebral control of the bladder in normal and urge-incontinent women. NeuroImage. 2007;37:1–7.

Bladder Aging

Vahabi B, Wagg AS, Rosier P, Rademakers KLJ, Denys MA, Pontari M, et al. Can we define and characterize the aging lower urinary tract? -ICI-RS 2015. Neurourol Urodyn. 2017;36:854–8.

Camões J, Coelho A, Castro-Diaz D, Cruz F. Lower urinary tract symptoms and aging: the impact of chronic bladder ischemia on overactive bladder syndrome. Urol Int. 2015;95:373–9.

Daly DM, Nocchi L, Liaskos M, McKay NG, Chapple C, Grundy D. Age-related changes in afferent pathways and urothelial function in the male mouse bladder. J Physiol. 2014;592:537–49.

Elbadawi A, Yalla SV, Resnick NM. Structural basis of geriatric voiding dysfunction. II. Aging detrusor: normal versus impaired contractility. J Urol. 1993;150:1657–67.

Dysfunctional Voiding

Peng CH, Chen SF, Kuo HC. Videourodynamic analysis of the urethral sphincter overactivity and the poor relaxing pelvic floor muscles in women with voiding dysfunction. Neurourol Urodyn. 2017;36(8):2169–75.

Artibani W, Cerruto MA. Dysfunctional voiding. Curr Opin Urol. 2014;24:330–5.

King AB, Goldman HB. Bladder outlet obstruction in women: functional causes. Curr Urol Rep. 2014;15(9):436.

Osman NI, Chapple CR. Fowler's syndrome—a cause of unexplained urinary retention in young women? Nat Rev Urol. 2014;11(2):87–98.

Brucker BM, Fong E, Shah S, Kelly C, Rosenblum N, Nitti VW. Urodynamic differences between dysfunctional voiding and primary bladder neck obstruction in women. Urology. 2012;80:55–60.

Groutz A, Blaivas JG, Pies C, Sassone AM. Learned voiding dysfunction (non-neurogenic, neurogenic bladder) among adults. Neurourol Urodyn. 2001;20:259–68.

Various

Cohn JA, Brown ET, Reynolds WS, Kaufman MR, Dmochowski RR. Pharmacologic management of non-neurogenic functional obstruction in women. Expert Opin Drug Metab Toxicol. 2016;12:657–67.

Costantini E, Lazzeri M, Bini V, Zucchi A, Fioretti F, Frumenzio E, et al. Open-label, longitudinal study of tamsulosin for functional bladder outlet obstruction in women. Urol Int. 2009;83:311–5.

Chapter 11
UDS in Pain Bladder Syndrome (PBS) and Overactive Pelvic Floor Dysfunction

11.1 Background

Bladder pain syndrome (BPS) is an enigmatic condition difficult to be diagnosed and treated. Its etiology and pathogenesis remain unknown, and a number of theories based on clinical and experimental observations have been developed without convincing evidence. Treatment strategies are empirical, with limited efficacy, and affected patients have a poor quality of life.

Much confusion regarding the diagnosis of pain bladder syndrome is due to many changes in definition and nomenclature since its first description in 1887 by Skene. The condition classically known as interstitial cystitis (IC) was reserved for patients with typical cystoscopic findings, such as glomerulations or the Hunner's ulcer (Fig. 11.1). Up until 2002, the National Institute for Diabetes and Digestive and Kidney Diseases (NIDDK) criteria, mostly based on glomerulations at cystoscopy under hydrodistention, were used to define IC.

Since NIDDK criteria were considered overly restrictive, in 2002 the ICS defined painful bladder syndrome (PBS) as "the complaint of suprapubic pain, related to bladder filling accompanied by other symptoms, such as increased daytime

G. Vignoli, *Urodynamics for Urogynecologists*,
https://doi.org/10.1007/978-3-319-74005-8_11

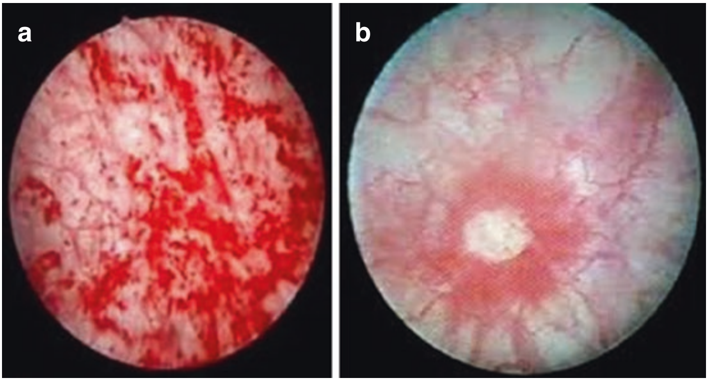


FIGURE 11.1 (**a**) Glomerulations are tiny areas of bleeding in the mucosa after bladder hydrodistention. (**b**) Hunner's ulcer is a mucosal lesion intermixed to normal capillary structure. Often it is covered by fibrin clots and associated with scars in the vicinity

and nighttime frequency, in the absence of proven urinary infection or other obvious pathology" of the lower urinary tract. Subsequent to this definition, some used IC to reflect patients who meet the classic NIDDK criteria and PBS to reflect those with identical symptoms but who did not undergo formal hydrodistention or did not meet all of the NIDDK criteria.

In the subsequent years, two more definitions have been proposed.

The ESSIC defined PBS as "chronic pelvic pain, pressure, or discomfort, perceived to be related to the urinary bladder accompanied by at least one other urinary symptom: persistent urgency or urinary frequency." This revised definition has been accepted by ICI.

The SUFU defined PBS as "an unpleasant sensation (pain, pressure, discomfort) perceived to be related to the urinary bladder, associated with lower urinary tract symptoms for more than six weeks duration, in the absence of infection or other identifiable causes." This definition is used by AUA.

There is a further Japanese and Asian view, in which urinary frequency/urgency is the primary symptom, with overactive bladder (OAB) and hypersensitive bladder (HSB) listed as subgroups and with painful bladder syndrome (PBS) as its extreme form (Fig. 11.2).

Beside terminology issues, 50–87% of patients suspected of having PBS manifest a voiding dysfunction due to an overactive pelvic floor.

Voiding dysfunction is defined by ICS as an intermittent and/or fluctuating flow rate due to an involuntary intermittent contraction of the peripheral striated muscle during voiding in neurologically normal individuals.

The overactive pelvic floor is "A condition in which the pelvic floor muscles do not relax, or may even contract when relaxation is functionally needed, for example during micturition or defecation" (2005 report from the Pelvic Floor Clinical Assessment Group of the ICS).

While in children this dysfunctional behavior may be the result of an improper learning of pelvic floor muscle control, in adults it is usually related to a voluntary prolonged holding

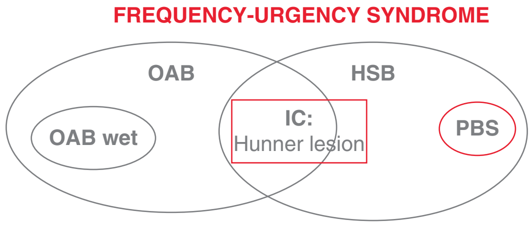


FIGURE 11.2 East Asian view of IC/PBS taxonomy. Symptoms of OAB and HSB substantially overlap. HSB is a condition of increased bladder sensation usually associated with urinary frequency and urgency with or without pain. The use of term "PBS/IC" is limited to patients with HSB symptoms and bladder pathology such as a Hunner's lesion or glomerulations after bladder overdistention. Controversial is the role of the finding of glomerulations following bladder hydrodistention since it has not proven to be of any significance

(habit, lifestyle, occupation, psychological distress) or may be a consequence of direct trauma of pelvic musculature (vaginal delivery, pelvic surgery).

Symptoms of overactive pelvic floor fall into three categories:

- Pain
- Bladder function
- Bowel function

In addition to voiding and defecation, other conditions such as sexual dysfunction and genital/pain syndromes (i.e., vulvodynia) are also commonly associated with pelvic floor muscle overactivity.

There is a very close relationship between overactive pelvic floor and chronic pelvic pain although the mechanisms of association are not completely understood. An overactive pelvic floor shares several mechanisms with myofascial pain syndrome. Myofascial pain syndrome typically occurs after a muscle has been contracted repetitively. The subsequent muscle spasm may induce the development of palpable taut bands and trigger points which are hypersensitive painful spots located within a definite set of tense muscle fibers (Fig. 11.3). In addition abnormal tension can cause the accumulation of inflammatory substances within the muscle

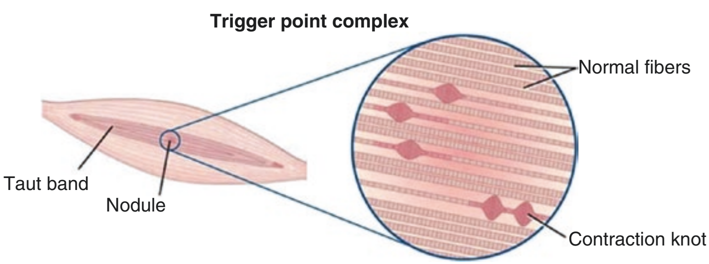


FIGURE 11.3 Trigger point complex. A hyperirritable spot in skeletal muscle that is associated with a hypersensitive palpable nodule in a taut band (group of tense muscle fibers extending around a trigger point)

which cause pain. Pain in turn increases muscle spam becoming a self-maintaining vicious cycle.

The role of pelvic floor overactivity is often neglected in the assessment of patients with pelvic problems despite the fact that a normalization of pelvic floor muscle function through physical therapy may be crucial for the successful management of symptoms.

11.2 Diagnosis of PBS/IC

Much work has been put into the attempt to define objective diagnostic criteria for PBS/IC based on cystoscopy during bladder hydrodistention, bladder wall biopsies, and urodynamics. Results have, however, been frustrating. A more advantageous approach is probably to establish a broad clinical diagnosis, mainly on the basis of symptoms and exclusion of other diseases, and then stratify patients by urodynamics, cystoscopy, histology, and other tests according to the significance of these findings regarding the treatment and prognosis of the disease.

Note: In PBS delay of diagnosis is common, with an average time of 3–7 years from the time of presentation to the general practitioner to diagnosis by a specialist.

11.2.1 Symptoms and Signs

The characteristic presentation of PBS/IC includes a combination of pain, frequency, nocturia, and urgency, with pain being the most common complaint. Typically pain, often described as "pressure," is felt in the suprapubic area, but it can be referred to other areas in the pelvis such as the urethra, vagina, labia, and perineum.

Worsening of symptoms ("flares"), triggered by stress, intercourse, menses, or diet are commonly reported. Frequency urgency is also prevalent and cannot distinguish PBS/IC from OAB. A poor response to antimuscarinics may suggest a

PBS. In addition, pain is usually relieved by micturition, which is not the case with OAB. Despite the absence of urinary infection (UTI) being a prerequisite at the time of diagnosis, up to 50% of patients will have a previous history of UTI.

A self-administered symptom score (also known as the O'Leary-Sant Symptom Score) may be useful to establish baseline symptoms severity.

The physical exam should include an abdominal and pelvic exam. A suprapubic tenderness particularly at the bladder neck level is often observed. In addition, palpating the anterior vaginal wall along the course of the urethra up to the bladder neck may elicit pain. Furthermore, in 50%–80% of the patients, a pelvic floor overactivity may be present. A systematic palpation of the pelvic floor muscles, looking for tenderness, spasm/taut bands, and/or trigger points, is important (Fig. 11.4).

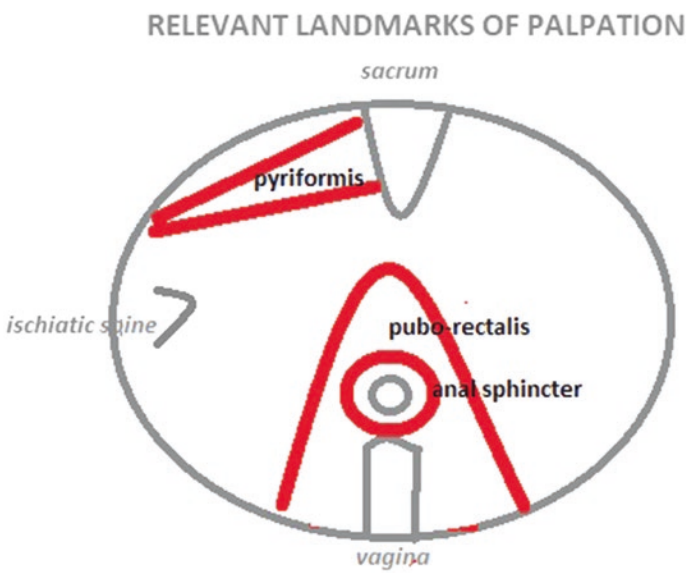


Figure 11.4 Pelvic floor muscles systemic palpation: relevant landmarks include anal sphincter, pubo-rectalis m. and pyriformis muscle

Also voiding diary helps to determine the severity of storage symptoms. Usually the average voided volume is less than 100 mL, and the average number of daytime voids ranges from 17 to 25 compared to 6 in an asymptomatic women. Pain should be recorded using a visual analogue scale (VAS) for pain during the 24 h of voiding diary.

A urine dipstick represents the minimum required laboratory test. If signs of UTI are identified, a culture and sensitivity are required.

11.2.2 Confusable and Associated Diseases

According to ESSIC, confusable disease is a disease which may show similar symptoms and/or signs to those of BPS/IC. In general, such a confusable disease needs to be excluded as the main cause of the symptoms and/or signs before a diagnosis of BPS can be made. Confusable diseases include malignancies, UTI, gynecological conditions, and other bladder conditions (see Box 11.1).

Box 11.1: PBS/IC—Relevant Confusable Diseases

Malignancies	*Other bladder conditions*
Bladder ca in situ	OAB-S
Cervical, uterine, ovarian cancer	VD-S
Urinary tract infections	*Gynecological conditions*
Intestinal bacteria	Endometriosis
Chlamydia, ureaplasma, mycoplasma	Candidiasis
Herpes simplex	POP
HPV	
Various	
Pudendal nerve entrapment	

Box 11.2: PBS/IC—Relevant Associated Diseases

- Allergic disorders
- Crohn’s disease
- Fibromyalgia
- Irritable bowel syndrome
- Rheumatoid arthritis
- Sjogren’s syndrome
- Lupus erythematosus
- Thyroid disorders
- Vulvodynia

Confusable diseases should be distinguished from *associated diseases*. Associated diseases are diseases with a higher prevalence among patients with BPS than in the general population. The practical consequence of associated diseases is that medical professionals should know these associations and should have a higher index of suspicion for PBS/IC.

Relevant associated diseases are reported in the Box 11.2.

11.2.3 Specific Tests

11.2.3.1 Cystoscopy

Cystoscopy performed alone, without hydrodistention, is expected to be normal (except for discomfort and reduced “functional” bladder capacity) in the majority of patients. The classic findings of glomerulations are reliably identified only after a formal hydrodistention under anesthesia. Therefore, the purpose of cystoscopy alone should only be viewed as a tool to rule out bladder cancer/carcinoma in situ.

11.2.3.2 Potassium Sensitivity Test

The potassium sensitivity test is no longer recommended as a standard evaluation for IC/BPS. The test is unreliable and mostly distressing, with patients experiencing severe pain both during and after the procedure.

11.2.3.3 Urodynamics

Overall, UDS studies are not recommended in the standard diagnostic evaluation of a patient suspected of having IC/BPS. Common findings on filling cystometry are a stable bladder with reduced first sensation of void (<100 mL) and reduced cystometric capacity (Fig. 9.5). Detection of detrusor overactivity, present in 15% of patients suspected of having IC/PBS, should exclude automatically the diagnosis, but may lead the clinician to initiate therapy with antimuscarinics. Whether these patients respond better to antimuscarinics than BPS patients with stable bladders has never been systematically investigated. Micturition assessment through pressure flow studies, with or without electromyography, may be useful in patients which complain coexistent voiding symptoms. In these cases a dysfunctional voiding due to an overactive pelvic floor is frequently seen (Fig. 10.1). Resting urethral profilometry usually indicate an high MUCP. Voiding dysfunction may be presumed less invasively by an intermittent "free" flowmetry pattern and by an ultrasound evaluation of post-void residual urine.

11.2.3.4 Hydrodistention

Hydrodistention under general anesthesia allows for stratification of patients into those with more classic disease associated with Hunner's lesion and glomerulations from those with no obvious mucosal abnormalities. The technique involves gravity filling of the bladder at 70–100 cmH_2O for a minimum of 2 min. Bladder capacity is fixed when the inflow backs up in the drip chamber or leakage occurs per urethra. A reduced

bladder capacity (< 400 mL) does correlate with pain, but more than 50% of patients with IC/BPS show capacities more than 800 mL under anesthesia. The presence of terminal hematuria upon draining the infusion fluid and the appearance of petechial submucosal hemorrhages (glomerulations) has been suggested to be characteristic of IC/BPS. A recent review of the literature, however, found no convincing evidence that glomerulations should be included in the diagnosis or phenotyping of bladder pain syndrome/interstitial cystitis. Glomerulations do not correlate with symptoms and are found in patients without bladder pain syndrome/interstitial cystitis. In addition, approximately 8% of patients with a diagnosis of IC/BPS do not show glomerulations. Hunner's ulcer seems more specific for IC. However, over the years, controversy has developed as to the prevalence and even the actual existence of the Hunner's lesion, and some urogynecologists believe that they are rare, or do not exist. Despite these limitations, hydrodistention is still the most accepted diagnostic criteria.

11.2.3.5 Bladder Biopsies

There are no specific features found on bladder biopsy to confirm a diagnosis of IC/BPS. Findings related to chronic inflammation are not specific, overlapping with other etiologies, and they correlate poorly to cystoscopic findings. In addition, between 30 and 43% of patients with a clinical diagnosis of IC/BPS may have normal histology.

Keynotes for Clinical Practice

- IC/PBS is a clinical and cystoscopic diagnosis.
- Urodynamic testing is not recommended for routine clinical use in IC/PBS diagnosis but only in patients refractory to first-line therapy.
- Pain during filling cystometry with a stable bladder (hypersensitivity) is consistent with IC/PBS.

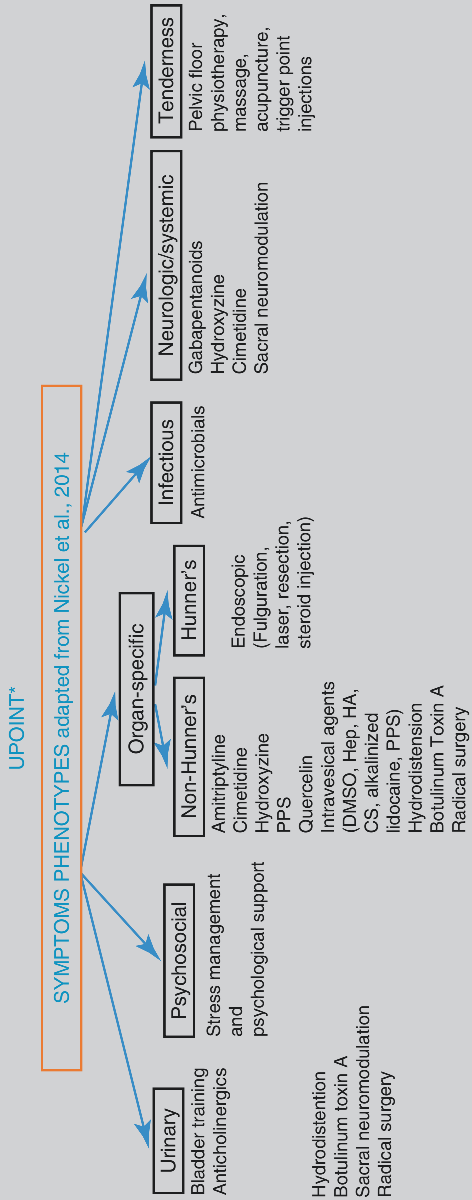
- Detrusor overactivity is seen in approximately 15–20% of IC/BPS patients. In these cases, it can be difficult to determine whether the diagnosis is DO alone or IC/BPS in combination with DO. Usually, patients with IC/PBS are less responsive to antimuscarinic drugs.
- Most patients will have normal compliance.
- Urodynamic evaluation may provide information regarding concomitant voiding dysfunction.
- Pelvic floor muscle dysfunction may manifest as intermittent flow and high resting urethral pressure.
- On pressure/flow study preferably with pelvic floor EMG, functional bladder obstruction can be diagnosed by a poor relaxing sphincter sometimes associated with poor detrusor contractility due to bladder inhibition from non-relaxing pelvic floor muscles.

11.3 Outline of Available Treatments

A complete discussion of the subject is beyond the goal of the chapter.

PBS/IC is a heterogeneous condition challenging to manage, with no treatment that is successful for all patients. Multiple options exist, ranging from conservative therapies with few side effects to major abdominal surgery. Patients can be identified with characteristic phenotypic patterns based on proposed mechanisms and symptom complexes. The UPOINT phenotypic classification system, recently described by Nickel and adopted by the EAU, may be a useful tool to characterize patients with PBS/IC and guide potential therapies (see Box 11.3).

Box 11.3: Phenotypic Management Paradigm for the Treatment of BPS/IC



*UPOINT (Urinary, Psychosocial, Organ-specific, Infective, Neurologic, Tenderness)

Suggested Readings

Overviews and Guidelines

Hanno P, Cervigni M, Dinis P, Nickel C, Nordling J, van Ophoven A, et al. Bladder pain syndrome. In: Abrams P, Cardozo L, Wagg A, Wein A, editors. Incontinence. Tokyo: ICS-ICUD; 2017. p. 2203–301.

Doggweiler R, Whitmore KE, Meijlink JM, Drake MJ, Frawley H, Nordling J, et al. A standard for terminology in chronic pelvic pain syndromes: a report from the chronic pelvic pain working group of the international continence society. Neurourol Urodyn. 2017;36:984–1008.

Cox A, Golda N, Nadeau G, Curtis Nickel J, Carr L, Corcos J, Teichman J. CUA guideline: diagnosis and treatment of interstitial cystitis/bladder pain syndrome. Can Urol Assoc J. 2016;10:E136–55.

Hanno PM, Erickson D, Moldwin R, Faraday MM, American Urological Association. Diagnosis and treatment of interstitial cystitis/bladder pain syndrome: AUA guideline amendment. J Urol. 2015;193:1545–53.

Davis NF, Gnanappiragasam S, Thornhill JA. Interstitial cystitis/painful bladder syndrome: the influence of modern diagnostic criteria on epidemiology and on Internet search activity by the public. Transl Androl Urol. 2015;4:506–11.

van de Merwe JP, Nordling J, Bouchelouche P, Bouchelouche K, Cervigni M, Daha LK. Diagnostic criteria, classification, and nomenclature for painful bladder syndrome/interstitial cystitis: an ESSIC proposal. Eur Urol. 2008;53(1):60–7.

Offiah I, McMahon SB, O'Reilly BA. Interstitial cystitis/bladder pain syndrome: diagnosis and management. Int Urogynecol J. 2013;24(8):1243–56.

Homma Y. Hypersensitive bladder: a solution to confused terminology and ignorance concerning interstitial cystitis. Int J Urol. 2014;21(Supp 1):43–7.

Cervigni M, Natale F. Gynecological disorders in bladder pain syndrome/interstitial cystitis patients. Int J Urol. 2014;21(Supp 1):85–8.

Fall M, Logadottir Y, Peeker R. Interstitial cystitis is bladder pain syndrome with Hunner's lesion. Int J Urol. 2014;21(Suppl 1):79–82.

Wennevik GE, Meijlink JM, Hanno P, Nordling J. The role of glomerulations in bladder pain syndrome: a review. J Urol. 2016;195:19–25.

Urodynamics

Kuo YC, Kuo HC. The urodynamic characteristics and prognostic factors of patients with interstitial cystitis/bladder pain syndrome. Int J Clin Pract. 2013;67(9):863.

Kirkemo A, Peabody M, Diokno AC, Afanasyev A, Nyberg LM Jr, Landis JR, et al. Associations among urodynamic findings and symptoms in women enrolled in the Interstitial Cystitis Data Base (ICDB) study. Urology. 1997;49:76.

Steinkohl WB, Leach GE. Urodynamic findings in interstitial cystitis. Urology. 1989;34:399.

Kuo HC, Chang SC, Hsu T. Urodynamic findings in interstitial cystitis. J Formos Med Assoc. 1992;91:694.

Various

Ogawa T, Ishizuka O, Ueda T, Tyagi P, Chancellor MB, Yoshimura N. Current and emerging drugs for interstitial cystitis/bladder pain syndrome (IC/BPS). Expert Opin Emerg Drugs. 2015;20:555–70.

Nickel JC, Irvine-Bird K, Jianbo L, Shoskes DA. Phenotype-directed management of interstitial cystitis/bladder pain syndrome. Urology. 2014;84:175–9.

Chapter 12
Neurogenic Lower Urinary Tract Dysfunction Syndrome (NLUTD-S) in Female

12.1 Background

Neurogenic bladder or neurogenic lower urinary tract dysfunction syndrome (NLUTD-S) is a bladder symptom complex caused by a lesion in the brain or spinal cord associated with a congenital condition (e.g., myelomeningocele), an acquired *stable* condition (e.g., stroke, spinal cord injury), or an acquired *progressive* condition (e.g., multiple sclerosis, Parkinson's disease, dementia).

The type of bladder dysfunction depends on the site, extent, and evolution of the lesion. From the functional point of view, the two most significant urodynamic findings are neurogenic detrusor overactivity (NDO), responsible of urinary incontinence, and detrusor sphincter dyssynergia (DSD), responsible of poor bladder emptying. Both result in elevated bladder pressure during the storage and voiding phases that often lead to structural bladder damage, vesicoureteral reflux (VUR), upper urinary tract dilation, and renal failure.

12.2 Classification

Several classification systems have been proposed. A simple classification system for use in daily practice that focuses mainly on therapeutic consequences has been proposed by

G. Vignoli, *Urodynamics for Urogynecologists*,
https://doi.org/10.1007/978-3-319-74005-8_12

Madersbacher and adopted by the EAU. This classification describes several NLUTD symptoms and findings on the basis of the contraction state of the bladder and external urethral sphincter during voiding and the filling phase (Fig. 12.1).

- Supraspinal lesion

Supraspinal lesions (above the pons) have predominantly storage symptoms, DO on urodynamics and absent PVR on ultrasounds.

- Spinal lesion

Spinal lesions (between the pons and sacral cord) have both storage and voiding symptoms, DO with DSD on urodynamics and PVR usually raised on ultrasounds.

- Sacral/infrasacral lesion

Sacral/infrasacral lesion (sacral cord and peripheral nerves) have predominantly voiding symptoms, hypocontractile or acontractile detrusor on urodynamics.

Note: Since bladders at the same level of lesion may behave differently over time depending on the etiology of neurogenic lesion, recently Powell proposed a new classification named SALE (Stratify by Anatomic Location and Etiology) to better guide the management of the bladder as well as inform about prognosis. The classification includes seven categories, each having a neurologic defect in a distinct anatomic location. In addition to bladder dysfunction, the presence or absence of bowel dysfunction and autonomic dysreflexia is reported. Furthermore, as more definite data will be gathered from the assessment of biomarkers, urinary nerve growth factor (NGF), and urinary brain-derived neurotrophic factor (BDNF), levels will be added to the classification to give more prognostic information.

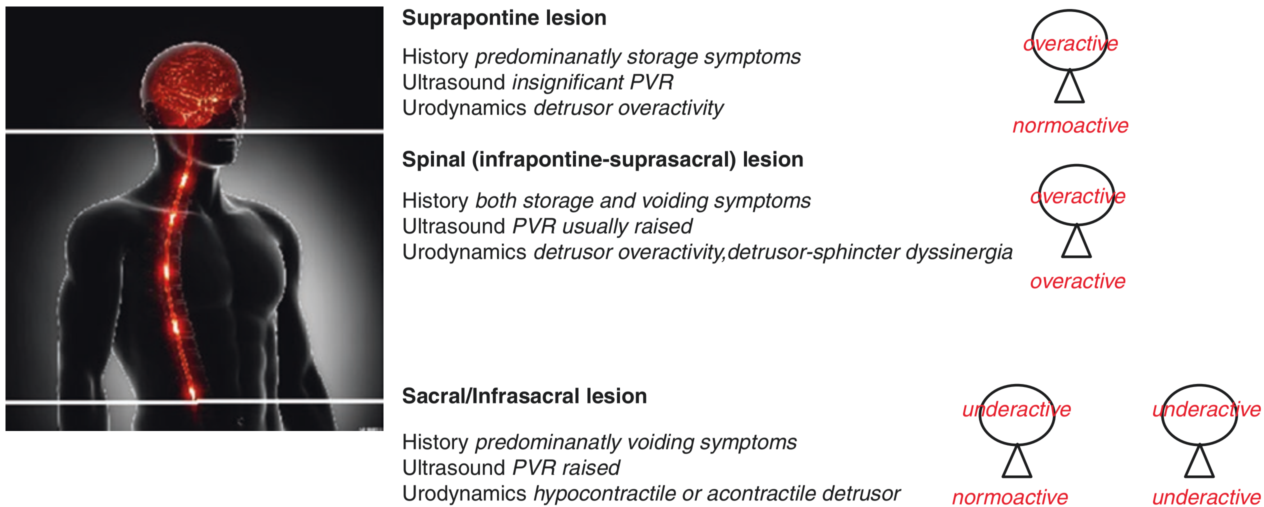


FIGURE 12.1 NLUTD classification

12.3 Clinical Assessment of Female with NLUTD-S

Despite the complexity of neural control, symptoms of neurogenic bladder dysfunctions can be simply classified in failure to store and failure to emptying with a large group of patients showing a combination of incomplete emptying and bladder overactivity.

Clinical assessment can be divided in:

- Initial assessment (noninvasive)
- Specialized assessment (mostly invasive)

12.4 Initial Assessment

12.4.1 History and Physical Examination

Neurologic history and neurological status including mobility, hand function, mental status, comprehension, spasticity, motor, and sensory status should be evaluated and prior surgery (hysterectomy, anti-incontinence surgery) and medications recorded. In parallel to the bladder symptoms, clinicians should ask about sexual and bowel dysfunction. Special attention should be paid to potential symptoms and signs that may indicate the presence of complications (i.e., pain, infection, hematuria, and fever). Quality of life should be assessed as well. Information obtained from the history can be supplemented by a bladder diary that records the time and volume of each voiding, urinary output and episodes of incontinence and urgency.

Physical examination should include the assessment of sensation in lumbosacral dermatomes, anal tone, and voluntary contraction of the anal sphincter, bulbocavernosus, and anal reflexes.

12.4.2 Routine Laboratory Investigations

Blood tests are performed to assess renal functions including serum creatinine. Urine dipsticks and, if necessary, urine culture are sent to exclude urinary tract infections, and this should also be carried out before invasive procedures such as urodynamics or cystoscopy.

Blood tests are performed to assess renal functions including serum creatinine. Urine dipsticks and, if necessary, urine culture are sent to exclude urinary tract infections, and this should also be carried out before invasive procedures such as urodynamics or cystoscopy.

12.4.3 Ultrasound

Ultrasound scanning can be used to assess morphological changes in the upper or lower urinary tract, such as upper urinary tract dilatation, stones, pyelonephritic scars, and, in advanced kidney disease, renal atrophy. Bladder ultrasound can detect stones, bladder wall thickness, and any large mucosal lesions. Bladder ultrasound is also performed in patients who can void, to assess the post-void residual (PVR) volume. In patients with impaired voiding, a PVR of more than 100 mL is thought likely to contribute to bladder dysfunction, although there is no consensus about the figure of PVR at which intermittent self-catheterization should be initiated.

Initial assessment will provide a presumed diagnosis of:

- Incontinence due to sphincteric weakness with negligible PVR
- Incontinence due to detrusor overactivity with negligible PVR
- Incontinence due to detrusor overactivity with associated emptying disorder (high PVR)

allowing an empirical treatment with intermittent catheterization and/or antimuscarinics alone or in combination.

12.5 Specialized Assessment

12.5.1 Urodynamics with Imaging Techniques

Often it is impossible to anticipate the nature of NLUTD-S from initial assessment alone, particularly in incomplete lesions. In addition, severity of NLUTD-S does not necessarily correspond with severity of neurological lesion. Despite a recent criticism, the use of multichannel cystometry and pressure flow studies with synchronous imaging (videourodynamics (VUD)) is considered the gold standard for the evaluation of patients with NLUTD-S.

Urodynamic investigation evaluates multiple functional parameters in NLUTD-S including:

- Bladder sensation during filling cystometry
- Detrusor function and compliance during filling cystometry
- Detrusor leak point pressure in patients with impaired detrusor compliance
- Cystometric bladder capacity
- Sphincter function during bladder filling
- Detrusor/sphincter function during voiding
- Post-void residual

Bladder pressure and sphincter EMG measurement, combined with fluoroscopy, are the ideal methods to investigate NLUTD-S. If videourodynamic facilities are not available, a simple cystography with voiding phase can be carried out. This will evaluate vesicoureteral reflux, indicate the level of bladder outlet obstruction (bladder neck, external urethral sphincter), and show the appearance of the bladder wall.

Compliance, detrusor leak point pressure, and "safe bladder capacity" are the most significant parameters recorded during filling phase. Detrusor leak point pressure is the lowest intravesical pressure at which leakage occurs around the catheter. The bladder volume at which detrusor pressure equals 40 cm H20 is considered patient's maximal "safe bladder capacity." Therefore, if the bladder volume remain below the maximal "safe bladder capacity," the detrusor pressure should remain safe for the upper urinary tract.

EMG of the urethral sphincter has been used for decades in the diagnosis of NLUTD-S; however, its value in practice remains uncertain. The use of needle or surface electrodes is debated. Urethral concentric needle electrodes were found to be superior to surface patch electrodes for evaluating relaxation of the muscle during voiding. Furthermore, with surface electrodes bladder dysfunction is related more to the anal sphincter than to the urethral sphincter. However, patch electrodes are more convenient for routine clinical practice.

Rhythmic detrusor contractions on cystomanometry with associated marked increase in EMG activity on attempted voiding are the relevant characteristics of patients with DSD. There are three types of DESD (Fig. 12.2). Type 1 has a crescendo increase in EMG activity that reaches a maximum at the peak of the detrusor contraction and then relaxes allowing unobstructed micturition, type 2 has clonic sphincter contractions interspersed throughout the detrusor contraction with a typically interrupted flow, and type 3 is characterized by a sustained sphincter contraction that

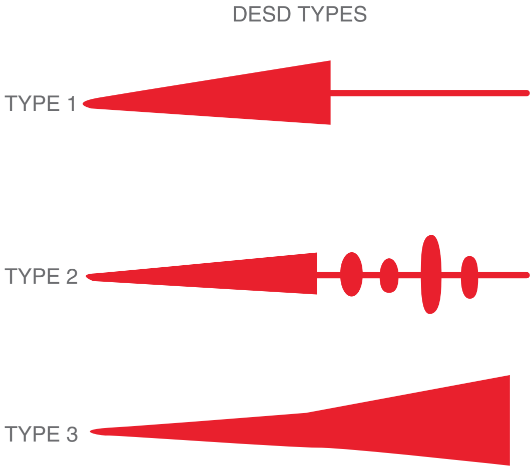


Figure 12.2 Types of DESD. Type 1 shows a temporary failure of the EMG to silence during detrusor contraction and is not obstructing. Type 2 is intermittent but not obstructing. Type 3 shows a crescendo of the EMG throughout the detrusor contraction and is obstructing

coincides with the detrusor contraction and does not permit any kind of flow. In some patients this high bladder pressure condition can give rise to autonomic dysreflexia (see below).

Note: A recent ICS report on terminology for adult neurogenic lower urinary tract dysfunction (ANLUTD) introduced two new types of neurogenic detrusor overactivity in addition to pre-existing phasic and terminal detrusor overactivity: *sustained detrusor overactivity*, defined as a continuous detrusor contraction whithout returning to the detrusor resting pressure and *compound detrusor contraction*, defined as a phasic detrusor contraction with subsequent increase in detrusor base pressure.

12.5.2 Difficult Situation During Urodynamic Testing in NLUTD-S: Autonomic Dysreflexia (AD)

Spinal cord injuries above the sixth thoracic neurotome (tetraplegia or high paraplegia) may be complicated by a phenomenon known as autonomic dysreflexia. AD is a potentially life-threatening acute condition due to excessive sympathetic responses to noxious stimuli below the level of lesion which leads to diffuse vasoconstriction and hypertension. A compensatory parasympathetic response produces bradycardia and vasodilation above the level of the lesion, but this is not sufficient to reduce elevated blood pressure. Typical stimuli include bladder distention and bowel impaction but also medical procedures such as urodynamic testing and cystourethroscopy. Common clinical manifestations of this troublesome event are headache, flushing, blurred vision, nausea, and increased blood pressure. Bradycardia is common.

Note: Patients with SCI above T6 have normal/baseline systolic blood pressure in the 90–110 mmHg range. Systolic blood pressure elevations more than 20 mm–40 mmHg above baseline may be a sign of AD.

Autonomic dysreflexia is the only urodynamic emergency. Management of acute attacks includes immediately sitting

the patient upright to orthostatically lower blood pressure, removal of any tight-fitting garments, correcting noxious eliciting stimuli (i.e., bladder distension), and prompt reduction of blood pressure with a rapid-onset/short-duration agent like nifedipine (10 mg SL) or captopril (25 mg SL).

Note: Unfortunately, it is not always possible to start the management on time, since the AD episode may be "silent", i.e., without clear symptoms. In these conditions, a further increase in blood pressure may end in a life-threatening condition. High index of suspicion is needed. Monitoring blood pressure during UDS testing is of utmost importance.

Avoidance of provoking stimuli is important in preventing attacks. Nifedipine, prazosin, and terazosin should be also used prophylactically to prevent an attack. Recently, a single cycle of 200 units of intradetrusor injected of onabotulinumtoxinA has been shown to decrease the severity and frequency of bladder-related AD episodes improving bladder function and quality of life.

12.5.3 Reappraisal of Urodynamic Testing and Videourodynamics in NLUTD-S

As mentioned before, the role of urodynamics and videourodynamics has been recently questioned since there are inadequate data to support its routine use in NLUTD-S. Overall, routine urodynamics should be recommended in conditions of "high-pressure bladder" as opposed to urodynamically "safe" bladder.

More specifically, the EAU recommend that in high-risk patients (defined by a high filling pressure (>40 cmH_2O), poor compliance (<10 mL/cm H_2O), and high detrusor leak point pressure (>40 cmH_2O)), upper urinary tract should be checked by urine laboratory, creatinine, and renal ultrasound once every 6 months (LE 4, Grade A) reserving conventional UDS or VUD when either are abnormal (*expert opinion*).

Likewise, the evidence regarding the added value of video to conventional urodynamics is controversial. In a broader context, VUD has been advocated in situations in which UDS

alone fails to provide sufficient diagnostic information to guide therapy, especially in patients with relevant neurological disease. Nevertheless, it is debated whether VUD is always necessary for every patient with NLUTD-S. As previously said, critical questions such as storage pressures and mechanisms of emptying can often be answered with simpler UDS testing eventually joined to a voiding cystourethrography (VCUG) if VUD facilities are unavailable. Current guidelines (ICS, ICI, AU, AUA/SUFU, NICE) do not specify the added value of imaging to urodynamics neither for management nor for outcome. In addition, none of these guidelines discusses the potential drawbacks (necessity of special equipment, time, and expense) and harm (radiation exposure) of the investigation. Recommendations are based on single center series and expert opinion.

12.6 Complications and Follow-Up of Neurogenic Bladder Dysfunction

Complications of neuropathic bladder dysfunction have been reported most often in patients with spinal cord injury. Up to the 1950s, upper urinary tract damage was the primary cause of mortality in this group of patients, but with newer advances, urinary complications are now relatively low. The commonest complications include upper and lower urinary tract infections and stones. Less commonly, morphological damage may occur in the lower urinary tract, such as diverticula, trabeculae, wall thickening, and pelvic organ prolapse. Upper tract complications include vesicoureteral reflux and kidney failure. In addition, the risk of squamous-cell carcinoma of the bladder is increased in patients with a long-term history of an indwelling catheter and recurrent urinary tract infections.

Patients with neurogenic bladder dysfunction, especially those with MS and spinal cord injury, need a regular neurourological follow-up. Optimum frequency and components of follow-up evaluations remain controversial. In spinal cord injury, most guidelines propose an assessment of the upper urinary tract functions using serum creatinine and ultrasound

every year reserving multichannel urodynamics to specific clinical situations like periodic control of high-pressure systems and uncontrollable UTI.

12.7 Principles of Treatment of NLUTD-S

The primary goals for treatment of NLUTD-S should be:

- Protection of the upper urinary tract
- Restoration of the LUT function (complete bladder emptying and dryness) and improvement of the patient's QoL

Note: The risk of developing upper urinary tract damage and renal failure is much lower in patients with slowly progressive non-traumatic neurological disorders than in those with spinal cord injury or spina bifida.

The principles of treatment of NLUTD-S are the same in female and male. However, practical management may be different between genders. Female patients usually have more severe urinary incontinence due to anatomical reasons but, unlike the male, cannot use external appliances to collect urine and prevent urine soiling. In addition, CIC may require more facilities and resources for women. It follows that women may need a larger amount of diapers to manage their incontinence and often recur to an indwelling catheter.

Restoration of LUT function includes:

- Management of bladder emptying
- Management of storage symptoms

Management typically begins with anticholinergic medications and clean intermittent catheterization. Patients who fail this treatment because of inefficacy or intolerability are candidates for a spectrum of more invasive procedures. Patients with incompetent sphincters are candidates for sling surgery or artificial sphincter implantation. Coordinated bladder emptying is possible with neuromodulation in selected patients. Bladder augmentation, usually with an intestinal segment, and urinary diversion are the last resort.

12.8 Management of Bladder Emptying

12.8.1 Spontaneous Voiding

Patients can be instructed to void by abdominal stimulation (triggered reflex voiding), the Crede maneuver, or abdominal straining (Valsalva). However, spontaneous voiding with and without triggered voiding and/or bladder expression has been proven to be less safe than CIC.

12.8.2 Intermittent Catheterization

Impaired emptying is most often managed by clean intermittent self-catheterization, and this should be initiated if the post-void residual urine is greater than 100 mL or exceeds one-third of bladder capacity or, rarely, if spontaneous voiding is dangerous due to high detrusor pressure.

Intermittent catheterization is designed to simulate normal voiding. Usually, the average adult empties the bladder four to five times a day. Thus, catheterization should be done four to five times a day; however, individual catheterization schedules may vary, depending on the amount of fluid taken in during the day.

CIC can be done by patient himself or by a caregiver.

The most common barriers on IC are insufficient hand function, inability to sit properly, and spasticity. IC may be easily applicable for patients with paired hand functions such as paraplegics, but it can be difficult for the patients who do not have enough hand functions. Unlike male, female patient presents several potential difficulties with IC in that the urethral meatus is more difficult to access particularly for patients confined to a wheelchair. In fact, the need to catheterize four to six times daily can be quite a challenge for many women confined to a wheelchair, since they need to recline or lie flat and remove clothing in order to access the urethral meatus and, unfortunately, in most cases, the accessible bathrooms are not large enough to recline or lie down.

In addition, for women who cannot successfully perform regular IC, there are no reliable devices to collect urine leakage. Therefore, many female patients confined to a wheelchair are more likely to use a chronic indwelling catheter or wear a diaper with all potential inherent dangers.

Fear of accidentally injuring self is another common concern among patients not confined to a wheelchair.

Appropriate instruction to the patient can help overcome natural reluctance (Fig. 12.3). Catheterization may be performed in any clean washroom, possibly with a counter space within reach. "Clean" technique, without disinfectant or gloves, usually prevents infection without the need for absolute sterility. Hands must be washed thoroughly with soap and water and then rinsed and dried. Fingernails should be short and clean. Position should be comfortable. At the beginning, a mirror could be helpful in outlining the anatomy. Vagina should be washed from front to back with soap and water. Catheters are usually pre-lubricated. Using nondominant hand (i.e., left hand for right-handed women), labia should be spread with index and ring fingers, whereas the tip of middle finger feel the urethral meatus. Then, using

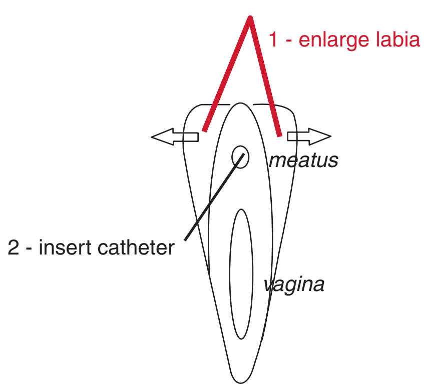


FIGURE 12.3 Technique of self-intermittent catheterization in women (see text)

dominant hand, catheter is slowly inserted into the urethra for about 5 centimeters until the urine begins to flow. Urine can be drained into a container or directly into the toilet if there is no need to measure the amount drained. Gentle straining may help bladder emptying. Catheter is then gently withdrawn (Box 12.1).

Box 12.1: Self-catheterization technique

1. Assemble the necessary supplies and have them close at hand.
2. Wash your hands with water and soap.
3. Get into a comfortable sitting position over the toilet rim or lying on the bed; then spread your thighs wide apart and separate vaginal labia with one hand.
4. Wash vaginal ostium from front to back with soap and water; then rinse and dry.
5. Squeeze the envelope of the catheter in order to lubricate it.
6. Using non-dominant hand spread labia to expose urethral opening; using the middle finger, feel the meatus.
7. Using dominant hand gently insert the catheter into the urethra for about 5 cm until the urine begins to flow.
8. Drain urine into a container or directly into the toilet if there is no need to know the amount of urine drained.
9. When urine stops flowing, withdraw the catheter slowly.

Another important concern about the IC is the infection. Although infection is a common problem in patients on IC, potential risk of infection is lower in patients on IC than in patients on indwelling catheterization. Usually, the infections that do occur are managed without complications. In general, routine use of long-term suppressive therapy with antibiotics in patients with chronic clean intermittent catheterization is not recommended since the use of chronic suppressive

antibiotic therapy may result in the emergence of resistant bacterial strains.

12.8.3 Indwelling Urethral Catheters

Many female patients prefer the simplicity of an indwelling catheter, and this view is also shared by many urologists. With an indwelling catheter, bladder pressures are not a problem, and the bladder is always adequately drained. However, an indwelling catheter is associated with significant potential morbidity including risks of recurrent urinary tract infection, hematuria, bladder stones, vesicoureteral reflux, and bladder cancer.

Indwelling catheters must be changed at least once a month. They may be changed at an office, a clinic, or at home by a visiting nurse. The standard catheter size is 16F or 18F, with a balloon filled with 10 mL of sterile water. Catheters that develop encrustations and problems with urine drainage must be changed more frequently. Routine irrigation of catheters is not required. However, some authors favor the use of 0.25% acetic acid irrigation (30 mL) because it is bacteriostatic, minimizes catheter encrustation, and diminishes the odor.

All indwelling catheters that remain in the urinary bladder for more than 2 weeks become colonized with bacteria. Bacterial colonization does not mean the patient has clinical bladder infection. When bladder infection occurs (foul odor, purulent urine, hematuria), the catheter and drainage system should be changed.

One of the major problems in females with an indwelling catheter is the erosion of the urethra. The typical presentation is that of a patient who has required a gradual increase in catheter diameter to stay dry with a progressive increase in the balloon fluid. Urethral erosion can be a very difficult problem that, invariably, requires surgical intervention. Reconstruction of damaged urethra with simultaneous autologous sling can be attempted with small urethral erosion and in patients willing to accept the practice of CIC after surgery. Otherwise, when urethral erosion is not amenable to sling placement, transabdominal bladder neck closure done in conjunction with a urinary diversion is a viable option.

12.9 Management of Storage Symptoms

Storage symptoms are most often managed using antimuscarinic medications. Other options include desmopressin to reduce urine output or intradetrusor injection of botulinum toxin type A to reduce detrusor overactivity.

12.9.1 Antimuscarinic Drugs

Anticholinergic treatment is the first-line therapy for neurogenic detrusor overactivity. This treatment works by blocking cholinergic transmission at muscarinic receptors. Several drugs are currently available including:

Anticholinergic agents have similar efficacy; however, they have different side effect and tolerability profiles that depend on their muscarinic receptor selectivity and the rate of drug distribution. Anticholinergic drugs that bind M1 receptors might produce impairments of memory and cognition. Agents that bind M2 receptors can produce QT interval prolongation that causes tachycardia and arrhythmias. Anticholinergic drugs that bind M3 receptors might produce constipation visual blurring and xerostomia.

The side effects of anticholinergic therapy have led to poor compliance with long-term use. In attempts to improve the efficacy and tolerability of anticholinergic therapy, several newer drugs have been developed that are available in sustained release formulations.

Sustained release formulations seem to have less side effects of the correspondent immediate release formulations. In addition, different routes of application have been proposed. A transdermal oxybutynin system was found to be efficacious in SCI patients with neurogenic detrusor overactivity and was well tolerated at up to three times the standard dose; however, skin irritation was a common side effect.

12.9.2 Botulin Toxin

Intradetrusor injections of onabotulinumtoxinA have transformed the management of neurogenic detrusor overactivity. Botulinum toxin A blocks neuromuscular transmission by binding to acceptor sites on motor or autonomic nerve terminals and inhibiting the release of acetylcholine. The inhibition occurs as the neurotoxin cleaves a protein critical to the successful docking and release of acetylcholine from presynaptic vesicles located within the nerve endings.

When injected into the muscle at therapeutic doses, botulinum toxin A produces a temporary chemical denervation of the muscle, resulting in a localized reduction in muscle activity. Evidence exists that reinnervation of the muscle may occur, thus slowly (6 months) reversing the muscle denervation produced by botulinum toxin A. Another mechanism for the effect of botulinum toxin A on detrusor overactivity may be related to its effect on sensory receptor expression in suburothelial fibers that may play a role in the pathophysiology of detrusor overactivity.

Botulinum toxin A may be used as monotherapy or in conjunction with anticholinergic therapy.

Intradetrusor injection of botulinum toxin A may be performed via rigid or flexible cystoscopy, depending on the urogynecologist's preference (Fig. 12.4).

The recommended dose is 200U per treatment, but doses of 150U and 100U achieve a similar rate of excellent results with significantly fewer adverse events including the duration of therapeutic effectiveness. The effects of treatments last at least 9 months.

The most common adverse event of intradetrusor injection of botulinum toxin A is an increased post-void residual, which may necessitate clean intermittent catheterization. Thus, patients should be counseled about the potential need for clean intermittent self-catheterization. Other side effects

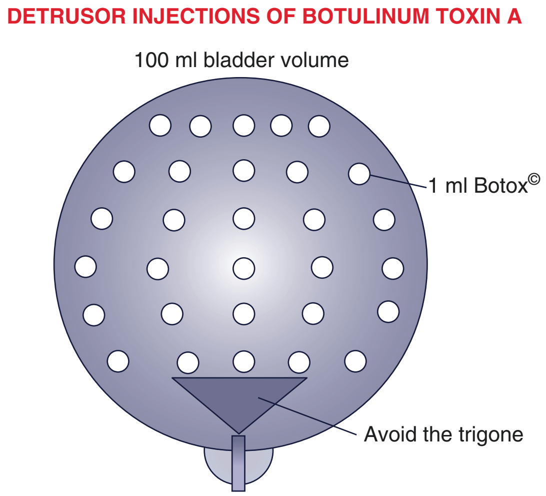


FIGURE 12.4 Schematic representation of intradetrusor injections of Botox (Allergan, Irvine, CA). At equally spaced points, several distinct injections according to the chosen dosage, each containing 1 mL of reconstituted onabotulinumtoxinA, are made sparing the trigone

include mild hematuria and urinary tract infection. Usually, there are no systemic side effects, but one study reported a mild case of asthenia after injection of botulinum toxin A 300U, which persisted for 10 days.

12.9.3 Neuromodulation

When pharmacotherapy fails to relax an overactive detrusor, neuromodulation may be considered a suitable option. Neuromodulation can be performed at different sites including sacral, pudendal, and tibial nerves. However, the most commonly described site of neuromodulation for treatment of OAB is the third sacral nerve root (S3).

Despite the substantial usage of the technique over the past two decades, the exact mechanism remains poorly

elucidated. The current leading hypothesis suggests that neuromodulation works by stimulating peripheral somatic afferent nerves (C fibers). Stimulation of the peripheral afferent nerve blocks competing abnormal visceral afferent signals from the bladder and prevents reflex bladder hyperactivity or retention However, the effects of neuromodulation are not just limited to spinal cord reflexes since functional magnetic resonance imaging (MRI) studies demonstrate changes of brain activity with SNM (Fig. 12.5).

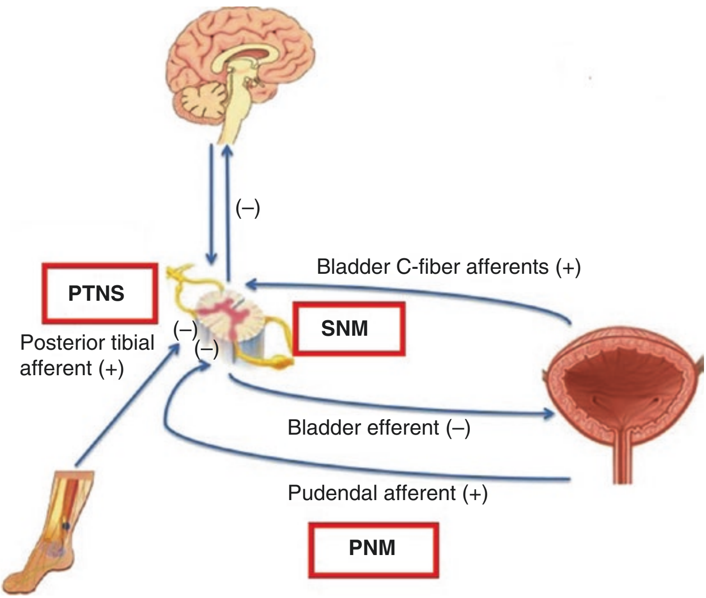


Figure 12.5 Techniques of neuromodulation. Neuromodulation is postulated to work in the setting of bladder overactivity by activating peripheral somatic afferent nerves (i.e., the pudendal or posterior tibial nerve) which in turn inhibit signals from the bladder afferents at the level of the spinal cord and thus disrupt an aberrant micturition reflex. Conversely, neuromodulation is postulated to work in the setting of urinary retention by restoring normal afferent signaling from the bladder in the midbrain and reducing cortical activity which stimulates the guarding reflex

Sacral and pudendal neuromodulation are performed as a staged procedure in order to demonstrate efficacy in the first phase prior to permanent implantation in the second phase. With PTNS the technique consists of stimulating the nerve by means of a 34 gauge needle electrode inserted 4–5 cm cephalad to the medial malleolus. Once the current is applied, the flexion of the big toe or the movement of the other toes confirms the correct positioning of the needle electrode. The electric current is a continuous, square wave form with a duration of 200 μs and a frequency of 20 Hz. The current intensity is determined by the highest level tolerated by the patient. The stimulation sessions last for 30 min and are performed once a week for 10–12 weeks in the majority of published papers.

Overall, neuromodulation techniques can only be used if there is little or no damage to the CNS-bladder axis. Data in the neurogenic population is limited to patients with stroke, Parkinson's disease, MS, and incomplete spinal cord injury. The current literature is generally positive and indicates that neuromodulation demonstrates a good efficacy among the neurogenic patients in terms of successful outcomes. However, the proportion of patients whose condition is improved is much less than in non-neurological pathologies. In addition, like in non-neurogenic patients, there is limited evidence for long-term efficacy (beyond 12–36 months), and maintenance of therapeutic effect requires reprogramming and surgical revision rate in a substantial number of patients.

Note: SNM is better utilized for treatment of bladder overactivity than for urinary retention. DESD is not an FDA-approved indication for SNM.

12.10 Surgery for Sphincteric Incompetence

12.10.1 Bulking Agents

Bulking agents can be used to treat neurogenic stress incontinence when there is a demand for a minimally invasive

treatment. The patient should be aware that the technique has a low success rate. Macroplastique (polymethylsiloxane) and Zuidex (dextranomer hyaluronic acid copolymer) are the most common synthetic products utilized. One of the main advantages of periurethral injections of bulking agents is that they are minimally invasive and can even be carried out as an outpatient procedure. The product is usually injected in the region of the bladder neck using a retrograde endoscopic approach. The bulking agent is injected at up to three sites with the aim of achieving urethral coaptation. The success rate, intended as "dryness for some hours" between voiding and catheterization, ranges between 0 and 36% with a mean follow-up that rarely exceeds 2 years.

12.10.2 Autologous Sling

In women with neurogenic lower urinary tract dysfunction, the use of an autologous fascial sling to support the urethra (usually close to the bladder neck) has been widely used to treat stress incontinence with a success rate of 83–89%. The procedure has a low morbidity rate. In particular, the risk of urethral erosion is very low when fascial slings are used.

After surgery, most of the patients will be dependent on bladder emptying using IC, because the patient's neurological dysfunction may preclude voluntary voiding and because the sling may have to be inserted under tension in order to provide sufficient outflow resistance in the face of neurogenic intrinsic sphincter deficiency.

Synthetic midurethral tapes have not been adopted for the treatment of neurogenic stress incontinence due to concerns about the risk of urethral erosion.

12.10.3 Artificial Sphincter

The AUS is recognized as one of the most effective treatments for urinary incontinence. It has the inherent advantage that it provides an adequate urethral closure pressure during

the urine storage phase allowing voiding to take place with a low bladder outlet resistance. In patients with SUI, it is able to deliver either complete or "social" continence in 75–87% with satisfaction rates ranging between 85 and 95%. Most of the series are related to male patients with non-neurogenic incontinence. Many teams are still reluctant to implant AUS devices in women because of technical difficulties related to the short length of female urethra, which can lead to subsequent complications. However, the device has been effectively used also in female patients with neurological disease provided that they had a low-pressure bladder. Data are scarce regarding the long-term functional outcomes of AUS among adult female neurological patients. In addition, the outcomes in the female neurological population, when available, are not analyzed separately from non-neurological patients. A thorough urodynamic assessment of the bladder is mandatory in order to evaluate the potential impact of bladder compliance following AUS implantation. In the event of any doubt about the quality of the bladder reservoir, bladder augmentation should be performed. Device infection, cuff erosion into the urethra, and loss of fluid from the implant are the main causes of failure of an AUS.

12.11 Diapers

Diapers are necessarily used in patients with storage symptoms when the aforementioned treatment regimens are not fully effective.

Diapers are pads designed to absorb urine to protect the skin and clothing. Usually available in disposable forms, they are a temporary means of keeping the patient dry until a more permanent solution becomes available or when the treatment gives less-than-optimal results.

By reducing wetness and odor, they help maintain the patient's comfort and allow them to function in normal activities. The improper use of pads may contribute to skin breakdown and urinary tract infections. Thus, appropriate use,

meticulous care, and frequent changes are needed when absorbent products are used.

With diapers it is important to implement a skin care program to avoid the development of moisture lesions. The aim is to keep the skin clean and dry. This can be achieved through the use of protective skin barrier products, gentle cleanser, and simple moisturizers. Following an episode of incontinence, the area should be cleaned using a foam cleanser (or similar). The skin should then be carefully dried, and either a moisturizer or skin barrier is applied depending on the condition of the skin and amount or frequency of incontinence. Skin care should routinely be done according to the degree of incontinence with timely cleansing of soiled and wet skin.

12.12 Types of Neurogenic Bladder and Relative Care

Some types of neurogenic bladder are common findings in urogynecological practice, while others are usually managed in high specialized centers. The first include dementia, stroke, multiple sclerosis, Parkinson's disease, diabetes mellitus, and radical pelvic surgery. The latter include spinal cord injury and spina bifida.

12.12.1 Dementia

Incontinence in dementia is complex because it is often multifactorial and does not lend itself to easy solutions. Detrusor overactivity (DO) was traditionally thought to be the principal cause of incontinence in dementia. However, much of the incontinence seen in dementia may be functional incontinence where the cognitive impairment interferes with the ability to toilet. Management is directed at ameliorating the predisposing causes with the ultimate goals of maintaining independence, self-esteem, and health of the person.

Anticholinergics have been used to reduce detrusor overactivity. However, the side effects of these drugs are a concern, as they can worsen cognition, raising the risk of delirium. More than drugs effective communication is the key to alleviating caregiving problems in dementia.

12.12.2 Stroke

Urinary incontinence following stroke is a common problem affecting more than one-third of acute stroke patients and persisting in up to a quarter at 1 year. It is well established that this condition is a strong marker of stroke severity and is associated with poorer functional outcomes and increased institutionalization and mortality rates compared with those who remain continent. Urodynamic findings vary depending upon timing of the study and associated comorbidities. Detrusor overactivity is predominant, and detrusor underactivity is somewhat less prevalent. Patients with detrusor underactivity should be managed with clean intermittent catheterization or indwelling Foley catheter, while timed voiding with or without anticholinergic therapy may be an effective treatment for patients with detrusor overactivity after stroke.

12.12.3 Multiple Sclerosis

Lower urinary tract dysfunction is a common problem in patients with multiple sclerosis (MS), with a prevalence of 75%. Urinary symptoms can vary in patients with MS, but usually the most common are urgency, frequency, nocturia, and urgency incontinence. In addition, patients can also present voiding symptoms, like hesitancy and intermittency, which can be present alone or in combination with the previous ones. This is well reflected by urodynamic findings, which shows that detrusor overactivity is the most common sign,

present in up to 81% of patients. The second most common finding is detrusor sphincter dyssynergia (DSD), which is associated to detrusor overactivity in 93% of cases. Treatment of detrusor overactivity in patients with MS is still based on anticholinergic drugs, after having assessed the post-micturition residual volume (PVR). However, anticholinergic drugs often fail to adequately control urinary symptoms or are abandoned due to side effects. Recently, the use of intradetrusor botulinum toxin type A, alone or associated to CIC, has proven to be effective in increasing continence, in improving urodynamic parameters, and in ameliorating quality of life.

12.12.4 Parkinson's Disease

Urinary disturbances are frequently observed in patients suffering of Parkinson's disease resulting in significant impact to the individual's quality of life. They may occur at any stage of the illness and get worse with advancing and aggravating disease. Storage symptoms are present in 57–83% of patients, whereas voiding symptoms are seen in 17–27% patients. Out of all the urinary symptoms, nocturia is the most common complaint in >60% patients with PD. Detrusor overactivity (DO) is the commonest urodynamic abnormality in patients with PD. The main differential to consider is multiple system atrophy (MSA) in which all patients are ultimately afflicted with urinary disturbance. It is well recognized that patients initially diagnosed with PD may in fact have MSA, and it is important to distinguish the two as their urological management is different. The post-void residual (PVR) volume is minimal in PD, which differs significantly from multiple system atrophy (MSA) patients who have a more progressive disease that leads to urinary retention. Anticholinergic drugs and, in selected cases, local injections of botulinum toxin are the mainstay of treatment.

12.12.5 Diabetes Mellitus

Over 50% women with diabetes of type 2 have bladder dysfunction. The incidence is correlated with the duration of diabetes and glycemic control.

The classic symptoms of diabetic cystopathy are decreased bladder sensation, increased bladder capacity, and impaired bladder emptying with resultant increased post-void residual volume. The syndrome, also known as diabetic cystopathy most likely represents end-stage bladder failure and is relatively uncommon. Recent clinical evidence indicates a prevalence of storage symptoms, such overactive bladder symptoms, in 39–61% of diabetic patients. The pathophysiology of diabetic cystopathy is multifactorial, including disturbances of the detrusor muscles, nerves, and urothelium. Hyperglycemia, oxidative stress, and polyuria play also important roles in inducing voiding dysfunction. A "temporal theory of diabetic bladder dysfunction" has been proposed. Hyperglycemia-induced polyuria plays a major pathophysiological role during the early stages of diabetes polyuria, causing compensatory bladder hypertrophy and associated myogenic and neurogenic alterations. This stage is compatible with findings of a hyperactive bladder during urodynamic evaluation when patients present with bladder storage concerns (urgency or urge incontinence). With time and accumulation of toxic metabolites, decompensation of bladder tissue and function ensues, resulting in the classical signs and symptoms of diabetic cystopathy (hypocontractile detrusor or atonic bladder) in patients with urinary voiding problems. The first line of treatment of urge incontinence is an oral anticholinergic drug. In case of empty failure, frequent clean intermittent catheterization is the best choice to avoid long-term indwelling catheter because of the risk of increased infection rate.

12.12.6 Radical Pelvic Surgery

Dysfunction of the lower urinary tract is the most common long-term complication of radical surgery for cervical cancer. Bladder disorders are related to the extent of radical surgery. The sympathetic and parasympathetic systems innervating the lower urinary tract may be disrupted due to resection of uterosacral and rectovaginal ligaments, the dorsal and lateral paracervix, the caudal part of the vesico-uterine ligaments, and the vagina. Nerve-sparing techniques appear to improve bladder function without compromising overall survival of the patients.

Typically there are two phases of dysfunction after surgery. In the immediate postoperative period, there is usually a transient spastic bladder with a decreased capacity but also with diminished sensation which may require urethral catheterization for a few days or for a few weeks. After this period the patient may recover an efficient voiding function, with some changes in bladder sensation and in detrusor and urethral function but without any symptoms. Permanent dysfunctions (beyond 6–12 months) are reported in 30–50% of patients. Hypocontractile bladder is the cause of persistent voiding dysfunction, and these patients need abdominal straining to void.

12.12.7 Spinal Cord Injury

Spinal shock occurs following an acute SCI and can last up to 3 months. Interruption of the nervous pathways eliminates the micturition reflex, the bladder becomes atonic, and there is no conscious awareness of bladder filling. The resulting urinary retention should be managed by clean intermittent catheterization (CIC) or with an indwelling catheter. Initial

urodynamic studies should be performed after the patient is beyond the spinal shock phase.

The subsequent evolution of bladder function will depend on the level of lesion:

- **In suprasacral lesion**, reflex bladder function will occur. Consciousness of bladder filling might not be totally absent; however, voluntary inhibition of the micturition reflex is lost. Typical urodynamic findings include detrusor overactivity and detrusor striated sphincter dyssynergia (DESD). Discoordinated micturition will result in urinary incontinence with high voiding pressure and residual urine volume, which, if not treated, will result in upper tract deterioration and renal failure.
- **Sacral lesions** will result in denervation of the bladder and sphincter. Micturition reflex is absent, and, in cases of complete lesion, conscious awareness of bladder filling is lost. Bladder is acontractile and usually highly compliant (in some cases low bladder compliance can occur) with competent but nonrelaxing smooth and striated sphincters that retain some fixed tone out of voluntary control.

12.12.8 Spina Bifida

Spina bifida, especially when it is associated with myelomeningocele, is the commonest congenital cause of neurogenic voiding dysfunction. More than 90% of children with spina bifida have bladder dysfunction. The most common finding is a urinary sphincter incompetence associated with bladder underactivity. However, detrusor overactivity can occur as well. The major problem with spina bifida is the risk of upper urinary tract damage which tends to increase with age. Adults

with spina bifida have eight times the age-standardized risk of renal failure compared to the general adult population.

Key Notes for Clinical Practice: Initial Assessment and Empirical Treatment of NLUTD-S

- History including of home circumstance
- Urinary diary
- *Physical examination*:
  - Assessment of mobility and hand function
  - Assessment of sensation in lumbosacral dermatomes, anal tone and voluntary contraction of anal sphincter, bulbocavernosus, and anal reflexes
- Urine analysis + culture
- Urinary tract imaging and serum creatinine
- PVR assessment

Presumed diagnosis according to level of lesion		
Suprapontine lesion (*Stroke, Parkinson's*)	Suprasacral lesion (*Spinal cord injury*)	Sacral cord/cauda equina lesion (*Disc prolapse, radical surgery*)
	Empirical Treatment	
Antimuscarinics[a]	Intermittent self-catheterizationa	Physical therapy[a]

Modified from Drake M, Apostolidis A, Cocci A, Emmanuel A, Gajewski JB, Harrison SC et al. Neurogenic lower urinary tract dysfunction: Clinical management recommendations of the Neurologic Incontinence Committee of the Fifth International Consultation on Incontinence 2013

[a]External pads and/or indwelling catheter can be used in support of the specific treatment

Key Notes for Clinical Practice: Specialized Assessment and Treatment of NLUTD-S

Urodynamic (usually videourodynamic) diagnosis			
Detrusor overactivity without DESD	Poor bladder emptying due to DESD	Poor bladder emptying due to detrusor underactivity	Sphincter incompetence
Conservative treatment			
AM + timed voiding	AM + IC	IC	None
	BonTA detrusor[a] + IC	Straining (?)	
	Triggered voiding (?) + alpha1-antagonists		
Surgical treatment			
Neuro-modulation	SDAF[b] + IC	None	Bulking agent
	SDAF[b] + SARS[c]		MUS (retropubic) + IC
	Enterocystoplasty + IC		Artificial sphincter

Modified from Drake M, Apostolidis A, Cocci A, Emmanuel A, Gajewski JB, Harrison SC et al.Neurogenic lower urinary tract dysfunction: Clinical management recommendations of the Neurologic Incontinence committee of the fifth International Consultation on Incontinence 2013

[a]BoNTA sphincteric injections are not currently licensed
[b]SDAF sacral deafferentation
[c]SARS sacral anterior-root stimulation

Note 1: Described by Brindley in 1970, SARS with posterior root rhizotomy can be performed only in patients with SCI with preserved sacral reflex and normal detrusor compliance. Because all or a part of the posterior sacral nerves are destroyed, the technique cannot be performed in patients with

conserved lower limb motility. Furthermore, the deafferentation induces sensory loss in the sacral dermatomes, and female patients could lose vaginal lubrification. Despite the efficacy, the technique is not commonly used because of the irreversibility and consequences of the posterior rhizotomy.

Note 2: Urinary diversion may be an option in selected cases of female patients with neurogenic bladder.

Note 3: Recent research suggest there are discrepancies between the goals of experts about how to best manage bladder problems in comparison to the goals of patients with neurogenic bladder. Obviously, nonadherence to codified programs can result in serious health problems (i.e., kidney stones, skin sores, urinary tract infection). On the other hand, proper adherence to bladder program may make it difficult to participate in social activities, especially for women. Updated clinical practice guidelines and standards for how to best assess and treat these problems in women are needed.

Suggested Readings

Overviews

Gajewsky JB, Schurch B, Hamid R, Averbeck M, Sakakibara R, Finazzi-Agrò E, et al. An International Continence Society (ICS) report on terminology for adult neurogenic lower urinary tract dysfunction (ANLUTD). Neurourol Urodyn. 2018;37:1152–61.

Drake M, Apostolidis A, Cocci A, Emmanue A, Gajewski JB, Harrison S, et al. Neurogenic lower urinary tract dysfunction: clinical management recommendations of the neurologic incontinence committee of the fifth international consultation on incontinence 2013. Neurourol Urodyn. 2016;35:657–65.

Panicker J, Fowler C, Kessler T. Lower urinary tract dysfunction in the neurological patient: clinical assessment and management. Lancet Neurol. 2015;14:720–32.

Goldmark E, Niver B, Ginsberg DA. Neurogenic bladder: from diagnosis to management. Curr Urol Rep. 2014;15:448.

Nambiar A, Lucas M. Chapter 4: guidelines for the diagnosis and treatment of overactive bladder (OAB) and neurogenic detrusor overactivity (NDO). Neurourol Urodyn. 2014;33(Suppl 3):S21–5.

Pannek J, Blok B, Castro-Diaz D, Del Popolo G, Kramer G, Radziszewski P, et al. EAU Guidelines on neurogenic lower urinary tract dysfunction. 2013.

Ginsberg D. The epidemiology and pathophysiology of neurogenic bladder. Am J Manag Care. 2013;19(10 Suppl):s191–6. Online.

Classification

Madersbacher HG. Neurogenic bladder dysfunction. Curr Opin Urol. 1999;9:303–7.

Powell C. Not all neurogenic bladders are the same: a proposal for a new neurogenic bladder classification system. Transl Androl Urol. 2016;5:12–21.

Assessment

Musco S, Padilla-Fernández B, Del Popolo G, Bonifazi M, Blok BFM, Groen J. Value of urodynamic findings in predicting upper urinary tract damage in neuro-urological patients: A systematic review. Neurourol Urodyn 2018, Feb 2. [Epub ahead of print]

Allio BA, Peterson AC. Urodynamic and physiologic patterns associated with the common causes of neurogenic bladder in adults. Transl Androl Urol. 2016;5:31–8.

Collins CW, Winters JC. AUA/SUFU adult urodynamics guideline: a clinical review. Urol Clin North Am. 2014;41:353–62.

Cruz F, Nitti V. Chapter 5: clinical data in neurogenic detrusor overactivity (NDO) and overactive bladder (OAB). Neurourol Urodyn. 2014;33(Suppl 3):S26–31.

Danforth TL, Ginsberg DA. Neurogenic lower urinary tract dysfunction: how, when, and with which patients do we use urodynamics? Urol Clin North Am. 2014;41(3):445–52.

Nitti VW. Evaluation of the female with neurogenic voiding dysfunction. Int Urogynecol J Pelvic Floor Dysfunct. 1999;10:119–29.

Management

Nevedal A, Kratz AL, Tate DG. Women's experiences of living with neurogenic bladder and bowel after spinal cord injury: life controlled by bladder and bowel. Disabil Rehabil. 2016;38:573–81.

Ginsberg DA. Management of the neurogenic bladder in the female patient. Curr Urol Rep. 2006;7:423–8.

Chartier-Kastler E, Denys P. Intermittent catheterization with hydrophilic catheters as a treatment of chronic neurogenic urinary retention. Neurourol Urodyn. 2011;30:21–31.

Jamison J, Maguire S, McCann J. Catheter policies for management of long term voiding problems in adults with neurogenic bladder disorders. Cochrane Database Syst Rev. 2013;11:CD004375.

Smith CP, Chancellor MB. Botulinum toxin to treat neurogenic bladder. Semin Neurol. 2016;36:5–9.

Kurpad R, Kennelly MJ. The evaluation and management of refractory neurogenic overactive bladder. Curr Urol Rep. 2014;15:444.

Cruz F, Herschorn S, Aliotta P, Brin M, Thompson C, Lam W, et al. Efficacy and safety of onabotu-linumtoxinA in patients with urinary incontinence due to neurogenic detrusor overactivity: a randomised, double-blind, placebo-controlled trial. Eur Urol. 2011;60:742–50.

Giannantoni A, Mearini E, Del Zingaro M, Porena M. Six-year follow-up of botulinum toxin a intradetrusorial injections in patients with refractory neurogenic detrusor overactivity: clinical and urodynamic results. Eur Urol. 2009;55:705–11.

Del Popolo G, Filocamo MT, Li Marzi V, Macchiarella A, Cecconi F, Lombardi G, et al. Neurogenic detrusor overactivity treated with english botulinum toxin a: 8-year experience of one single centre. Eur Urol. 2008;53:1013–9.

Karsenty G, Denys P, Amarenco G, De Seze M, Gamé X, Haab F, et al. Botulinum toxin A (Botox) intradetrusor injections in adults with neurogenic detrusor overactivity/neurogenic overactive bladder: a systematic literature review. Eur Urol. 2008;53:275–87.

Church B, Denys P, Kozma CM, Reese PR, Slaton T, Barron RL. Botulinum toxin A improves the quality of life of patients with neurogenic urinary incontinence. Eur Urol. 2007;52:850–8.

Sanford MT, Suskind AM. Neuromodulation in neurogenic bladder. Transl Androl Urol. 2016;5:117–26.

Joussain C, Denys P. Electrical management of neurogenic lower urinary tract disorders. Annals Physical Rehab Med. 2015;58(4):245–50.

Lay AH, Das AK. The role of neuromodulation in patients with neurogenic overactive bladder. Curr Urol Rep. 2012;13:343–7.

Cameron AP. Medical management of neurogenic bladder with oral therapy. Transl Androl Urol. 2016;5:51–62.

Abdul-Rahman A, Attar KH, Hamid R, Shah PJ. Long-term outcome of tension-free vaginal tape for treating stress incontinence in women with neuropathic bladders. BJU Int. 2010;106:827–30.

Pannek J, Bartel P, Gocking K. Clinical usefulness of the transobturator sub-urethral tape in the treatment of stress urinary incontinence in female patients with spinal cord lesion. J Spinal Cord Med. 2012;35:102–6.

Phè V, Leon P, Granger B, Denys P, Bitker MO, Mozer P, et al. Stress urinary incontinence in female neurological patients: long-term functional outcomes after artificial urinary sphincter (AMS 800TM) implantation. Neurourol Urodyn. 2017;36:764–9.

Averbeck M, Madersbacher H. Follow-up of the neuro-urological patient: a systematic review. BJUI. 2015;115:39–46.

Specific Types of NLUTD

Ransmayr GN, Holliger S, Schletterer K, Heidler H, Deibl M, Poewe W, et al. Lower urinary tract symptoms in dementia with Lewy bodies, Parkinson disease, and Alzheimer disease. Neurology. 2008;70:299–303.

Mehdi Z, Birns J, Bhalla A. Post-stroke urinary incontinence. Int J Clin Pract. 2013;6:1128–37.

Winge K. Lower urinary tract dysfunction in patients with parkinsonism and other neurodegenerative disorders. Handb Clin Neurol. 2015;130:335–56.

Phè V, Chartier-Kastler E, Panicker J. Management of neurogenic bladder in patients with multiple sclerosis. Nat Rev Urol. 2016;13:275–88.

Laterza RM, Sievert KD, de Ridder D, Vierhout ME, Haab F, Cardozo L, et al. Bladder function after radical hysterectomy for cervical cancer. Neurourol Urodyn. 2015;34:309–15.

Daneshgari F, Liu G, Birder L, Hanna-Mitchell AT, Chacko S. Diabetic bladder dysfunction: current translational knowledge. J Urol. 2009;182(6 Suppl):S18–26.

Kirschner-Hermanns R, Daneshgari F, Vahabi B, Birder L, Oelke M, Chacko S. Does diabetes mellitus-induced bladder remodeling affect lower urinary tract function? ICI-RS 2011. Neurourol Urodyn. 2012;31:359–64.

Taweel WA, Seyam R. Neurogenic bladder in spinal cord injury patients. Res Rep Urol. 2015;7:85–99.

Mourtzinos A, Stoffel JT. Management goals for the spina bifida neurogenic bladder: a review from infancy to adulthood. Urol Clin North Am. 2010;37(4):527–35.

Chapter 13
Making Sense of Urodynamic Testing for Women with LUTS: Practical Conclusive Notes

The enthusiasm of routine urodynamic testing in the management of women with lower urinary tract dysfunction, in particular urinary incontinence and pelvic organ prolapse, has been tempered in the last few years. Controversy exists whether routine urodynamic testing provides a real benefit for the patient since there is a growing evidence that they give little additional information over basic office assessment, including focused history with quality of life (QoL) assessment, bladder diaries, pelvic examination, cough test, uroflowmetry, measurement of post-void residual urine, and urine analysis.

Clearly, the exclusion of preoperative urodynamics in favor of comprehensive office evaluation in women with SUI or stress-predominant MUI might lead to significant savings for healthcare systems and could also save women from the embarrassment of the examination and from potential urinary tract infections. In addition, some reports demonstrated that immediate sling placement for women with pure SUI or MUI was less costly and more effective than a delayed treatment based on UDS finding.

The problem turn around the distinction between pure stress urinary incontinence and complicated urinary incontinence with mixed urinary incontinence staying between the two.

G. Vignoli, *Urodynamics for Urogynecologists*,
https://doi.org/10.1007/978-3-319-74005-8_13

The National Institute for Health and Care Excellence (NICE) guideline recommends that urodynamics not be performed in the *small* group of women where pure SUI is diagnosed based on a detailed clinical history and physical examination.

ICS defines complicated urinary incontinence as incontinence with significant pelvic organ prolapse, recurrent infections, pain or hematuria, suspected or proven voiding problems, failed previous incontinence surgery, or incontinence following radical pelvic surgery. This group of women with complicated UI will still need urodynamics.

Mixed urinary incontinence, highly prevalent among elderly patient, is a challenge. Women with mixed symptoms may present underlying pure detrusor overactivity or different combinations of stress incontinence with detrusor overactivity, urethral hypermobility, and intrinsic sphincteric deficiency with or without voiding dysfunction.

The relationship between SUI and the overactive bladder (OAB) syndrome is still poorly understood. At least 50% of patient could benefit from treatment directed at the urethra, but the other 50% could complain a worsening of symptoms. Given that patients undergoing stress incontinence surgery expect not only the resolution of urine leakage but also urgency and frequency symptoms and given that these symptoms are among those that predict postsurgery dissatisfaction, UDS has a pivotal role in defining detrusor function not only in terms of the presence or absence of detrusor overactivity but also in terms of increased afferent activity and brain handling of peripheral bladder informations.

The question remains whether the overall outcome of surgery could have a better success rate when a more structured UDS diagnosis had been used.

Beyond precluding indication to surgery, urodynamic information can facilitate tailored counseling of patients regarding the need for postoperative treatment for urgency/urgency incontinence.

Currently, there is no adequate consensus on how to categorize SUI in terms of the two principal postulated

pathophysiological mechanisms: intrinsic sphincter deficiency and urethral hypermobility. These represent extremes of a spectrum and coexist in the vast majority of patients. The uncertainty over this categorization is recognized by the ICS, which has called for further research in this area.

Despite abdominal leak point pressure or maximum urethral closure pressure does not seem to alter the surgical outcome of a MUS, a retropubic tape appears to be more suitable than a transobturator tape in women with intrinsic sphincter deficiency, diagnosed using urethral pressure profilometry and/or abdominal leak point pressures.

In the case of pelvic organ prolapse, UDS testing should clarify which patients might have lower urinary tract dysfunction following repair, i.e., incontinence, OAB symptoms, and voiding dysfunction. Clinical testing would seem easier for most patients and physicians, but it is conceivable that UDS testing may offer the most efficient way to identify the relevant information necessary to make a clinical decision. Once again, it should be acknowledged that particularly for OAB symptoms and voiding dysfunction, the final decision will be no better than without UDS testing and the results, in fact, will be used only to counsel the patients about the possible persistence of symptoms following surgery.

Voiding dysfunction in female is a relatively new field. Mechanical obstruction, detrusor underactivity, and overactive pelvic floor are the acknowledged underlying mechanisms. Mechanical obstruction, although the intriguing difficulties are associated with the unclear definition of obstruction in female, is probably the easier urodynamic diagnosis.

Poor detrusor contractility is an aging-related problem that may be associated with a postoperative voiding dysfunction that may create significant care problems in elderly patients.

The mechanisms are still poorly understood. However, identifying patients likely to develop postoperative VD may be useful in better defining patient expectations and identifying those most likely to benefit from preoperative teaching of clean intermittent self-catheterization.

Finally, the recognition of pelvic floor overactivity may be a key element to address patient to physical therapy instead of surgery.

In summary, the use of urodynamics depends on whether the clinician feels that the extra information provided by the investigation translates into better outcomes and so would be worth the associated risks and costs of the examen. From urodynamic perspective the pathophysiology of symptoms is multifactorial in women with LUTS, and UDS testing is the only objective way to determine the underlying mechanisms.

Irritative symptoms and emptying problems are probably the clinical situations that take the best advantage from urodynamic examination. Cystometry is the only method by which bladder sensations and control can be evaluated, and pressure-flow study is the only proven method to evidentiate voiding problems other than obstruction, i.e., detrusor underactivity and pelvic floor overactivity. UDS add a dimension of precision to evaluation of female with LUTS and sometimes, according to the current views, may significantly influence technique and outcome measures in this group of patients. Despite the passionate debate, many clinicians still perform preoperative multichannel UDS. A recent survey of urologists and urogynecologists concluded that many of them would arrange urodynamics for women with LUTS prior to surgery while recognizing the need for further better designed clinical trials to address this question.

Suggested Reading

Hilton P, Bryant A, Howel D, McColl E, Buckley BS, Lucas M, et al. Assessing professional equipoise and views about a future clinical trial of invasive urodynamics prior to surgery for stress urinary incontinence in women: a survey within a mixed methods feasibility study. Neurourol Urodyn. 2012;31:1223–30.

Appendix A: What Do the Experts Say?—Scientific Societies Recommendations for UDS Testing

Acronyms

AHPCR	Agency for Healthcare Policy and Research
SOGC	The Society of Obstetricians and Gynecologists of Canada
ACOG	The American College of Obstetricians and Gynecologists
ICS	International Continence Society
RCOG	Royal College of Obstetricians and Gynecologists
NICE	National Institute of Clinical Excellence
EAU	European Association of Urology
ICI	The International Consultation on Incontinence
AUA	American Urological Association
SUFU	Society of Urodynamics, Female Pelvic Medicine & Urogenital Reconstruction

Numerous scientific societies have provided opinions on the indications for urodynamic testing. Below are summarized the main clinical guidelines.

- AHPCR

Multichannel urodynamic testing should be reserved only for women with "complicated diagnostic situations or involved therapeutic plans."

Fantl JA, Newman DK, Colling J, DeLancey JOL, Keeys C, Loughery R, et al. Urinary incontinence in adults: acute and

G. Vignoli, *Urodynamics for Urogynecologists*,
https://doi.org/10.1007/978-3-319-74005-8

chronic management. Rockville, MD. Department of Health and Human Services (US), Public Health Service, Agency for Health Care Policy and Research, 1996. Clinical practice guideline N0. 2, AHPCR publication No. 96–0682 24.

- RCOG

Prior to performing irreversible bladder neck surgery, it would appear to be beneficial to have assessed objectively the type of incontinence and the presence of any complicating factors.

Adams EJ, Barrington JW, Brown K, Smith ARB. Surgical Treatment of Urodynamic Stress Incontinence. Royal College of Obstetricians and Gynaecologists Guideline No. 35, October 2003.

- SOGC and ACOG

Preoperative urodynamic testing are not necessary in women with pure stress incontinence that can be objectively demonstrated in whom all appropriate preoperative investigations have been performed.

Farrell SA. SOGC Urogynaecology Committee. The evaluation of stress incontinence prior to primary surgery. SOGC Clinical Practice Guideline No. 127. J Obstet Gynaecol Can. 2003;25(4):313–24.

American College of Obstetricians and Gynecologists. Urinary incontinence in women. ACOG Practice Bulletin No. 63. Obstet Gynecol. 2005;105:1533–45.

- ICS

Non-invasive urodynamics, such as voiding diary, post-void residual, and uroflowmetry, are recommended for all incontinent patients.

Invasive urodynamic studies are not necessary prior to treatment when the type of incontinence is clear and there are no complicating factors involved.

Abrams P, Cardozo L, Khoury S, Wein A. Incontinence, Basics and Evaluation, 3rd International Consultation on Incontinence, Co-sponsored by the International Continence Society, Edition 2005.

- ICI

UDS should be used if results will alter treatment recommendation and management.

Abrams P, Andersson KE, Birder L, et al. Fourth International Consultation Incontinence Recommendations of the International Scientific Committee: evaluation and treatment of urinary incontinence, pelvic organ prolapse, and fecal incontinence. Neurourol Urodyn. 2010;29:213–40.

- AUA/SUFU

Urodynamics may be "optionally" performed in patients with UI if considering invasive treatment.

Appell RA, Dmochowski RR, Blaivas JM, et al. Guideline for the Surgical Management of Female Stress Urinary Incontinence: 2009 Update. American Urological Association Education & Research, Inc, 2012 revision.

- NICE

Urodynamics is unnecessary in case of pure SUI.

Urodynamic testing should be considered if diagnosis is unclear, with a history of previous surgery for SUI or for symptoms suspicious for detrusor overactivity or voiding dysfunction.

National Institute for Health and Care Excellence. Urinary Incontinence in Women: The Management of Urinary Incontinence in Women, 2013.

- Cochrane

While urodynamic tests may change clinical decision making, there is some evidence that this do not result in better outcomes in terms of a difference in urinary incontinence rates after treatment.

Clement KD, Lapitan MC, Omar MI, Glazener CM. Urodynamic studies for management of urinary incontinence in children and adults. Cochrane Database Syst Rev. 2013.

- EAU

Urodynamics is unnecessary if pursuing a conservative treatment.

UDS should be used if results may alter treatment recommendation and management.

Counseling the patient that UDS does not predict the treatment outcome is advisable.

Test of urethral function by urethral pressure profile or leak-point pressure measurement should not be used.

Burchard F, Bosch J, Cruz F, et al. EAU Guidelines on Urinary Incontinence. Updated March 2017.

Suggested Reading

Syan R, Brucker B. Guidelines of guidelines: urinary incontinence. BJU Int. 2016;117:20–33.

Appendix B: Informed Consent for Invasive UDS Testing (Sample)

Definition

Urodynamic testing is a sophisticated office-based procedure used to help diagnose problems with difficult urination and/or involuntary loss of urine. Urodynamic testing helps increase the accuracy of diagnosis, uncovering in some instances a completely unexpected finding. The test is short and minimally invasive. In certain circumstances, even more information can be obtained with the use of fluoroscopy (special real-time X-rays) during the test. This is referred to as a "videourodynamic test."

Preparation

There is no particular preparation for a UDT. If you are in child-bearing age, it is important that you are not pregnant especially in cases where the use of X-ray is planned. So please inform the examiner if there is any suspicion that you may be. You should arrive to the office with a full bladder in order to perform an uroflowmetry. Uroflowmetry is the initial portion of the test in which you urinate into a special equipment that calculate the pattern and the force of your stream.

Procedure

The examen will take less than 1 hour.

Once your bladder is empty, you will be asked to lie down on the examination table. Under sterile conditions a small catheter will be inserted in your bladder through the urethra. With the insertion you may feel a minimal discomfort. Next, a similar catheter with a small balloon on the end will be inserted into the rectum or the vagina, and the balloon will be filled with a little amount of fluid. The catheters will be secured in place with a tape, the bed will be put in sitting position, and the test will begin. Throughout the filling, you will be asked to describe the sensations you are having as the bladder fills. When you are full, you will be asked to urinate and empty your bladder into a special container. In certain instances, X-rays can be taken during the filling and voiding portions of the test. In these cases, bladder will be filled with an X-ray dye instead of saline solution. Don't worry about possible allergies to dye because the fluid is only in your bladder and not in your bloodstream. At the end of voiding, catheters are removed and the test is over.

Post-procedure

After the procedure you might have a little burning in the urethra that usually disappear with the following urination. You may even see a tiny blood discoloration of your urine.

Possible Complications of the Procedure

All invasive procedures, regardless of complexity or time, can be associated with unforeseen problems. These include:

Urinary Tract Infection

It may be a simple bladder infection that presents with symptoms of burning urination, urinary frequency, and a strong urge to urinate and usually resolve with a few days of antibiotics.

Bloodstream Infection (Urosepsis)

This type of infection often presents with the urinary symptoms and any combination of the following: fevers, shaking chills, weakness or dizziness, nausea, and vomiting. You may need a short hospitalization for intravenous antibiotics, fluids, and observation. This scenario is more common in diabetics, patients on long-term steroids, or patients with any disorder of the immune system.

If you have high temperatures or any symptoms of severe illness (fevers, shaking chills, weakness or dizziness, nausea and vomiting, confusion), let your doctor know immediately, or proceed to the nearest emergency room.

Blood in the Urine

In some patients, placing the catheters within the bladder may cause a very small amount of bleeding in the urine. In almost all instances, the urine clears on its own over the next day or so.

Consent for Treatment

I understand that during the course of the procedure, unforeseen conditions might arise that could require other operations, procedures, or treatments.

I acknowledge that the procedure has been explained to me in detail and all my questions concerning the procedures have been answered to my satisfaction.

I hereby consent to the above procedure accepting all of the inherent risks.

Patient signature Physician signature

Index

G. Vignoli, *Urodynamics for Urogynecologists*,
https://doi.org/10.1007/978-3-319-74005-8